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A practical treatise on medical diagnosis



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A PRACTICAL TREATISE
ON
MEDICAL DIAGNOSIS
FOR STUDENTS AND PHYSICIANS

BY
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SIXTH EDITION, REVISED

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HOSPITAL OF THE UNIVERSITY OF PENNSYLVANIA

ILLUSTRATED WITH 196 ENGRAVINGS AND 27 COLORED PLATES



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PREFACE TO SIXTH EDITION

IN the preface to the fifth edition, it was stated that the original purpose of the work was "to make it an exponent of objective medicine and to point out the way to acquire precision in diagnosis." In revising the work of my father, Dr. J. H. Musser, I have carefully endeavored to keep this purpose before me, preserving the splendid store of clinical material in the book and bringing it abreast of our knowledge of today.

While I have carefully retained the plan and methods pursued in preparing the last edition, the enormous advances in medical science during the past eight years made it imperative that the book should be practically rewritten. This necessity rendered it possible, by rearrangement and by judicious condensation, to make the present edition of a more convenient size, while it is believed that none of those factors to which the great popularity of the earlier editions was due, have been sacrificed.

In the sections on the infectious diseases, the diseases of the cardiovascular system, the metabolic diseases, the diseases of the gastrointestinal and the urinary systems, much new material has necessarily been incorporated on account of the many recent additions to our knowledge of the causes and manifestations of these conditions. The chapter on the infectious diseases has been divided into two parts, the first containing those diseases due to vegetable organisms, the second those due to animal parasites. New sections have been added to the discussion of the disturbances of the internal secretions. A new chapter has been added dealing with the various functional tests of organic efficiency that have proved of such value in diagnosis and prognosis in the last few years. The sections on laboratory diagnosis have been extensively revised, and only those important tests have been inserted which may be performed in such a clinical laboratory as every physician could and should possess. In the section on physical diagnosis many changes have been made, particularly in the division dealing with the graphic methods of diagnosing cardiac disorders, and new illustrations have been added representing the various types of arrhythmia.

The first portion of the book containing the sections on the history and the symptoms of disease has been abbreviated, but it is believed that all the essentials have been retained, while much new diagnostic data have been added.

It is hoped that the condensation will prove of value to the student, as well as to the practising physician, as the important and essential facts are discussed with but a minimum amount of necessary explanation. This has required, at times, a somewhat didactic statement of the facts.

My thanks are due to the publishers, Messrs. Lea & Febiger, for the material assistance and courtesy rendered me in the performance of a difficult undertaking.

J. H. M., JR.

PHILADELPHIA, 1913.

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MEDICAL DIAGNOSIS

PART I GENERAL DIAGNOSIS

SECTION I GENERAL CONSIDERATIONS. HISTORICAL DIAGNOSIS. DATA OBTAINED BY INQUIRY

CHAPTER I

THE DATA, METHODS, AND OBJECTS OF DIAGNOSIS

THE sufferings of one who consults a physician are indicated by *symptoms*. The symptoms of which the patient complains are known as the *subjective symptoms*. The symptoms which the physician observes are known as the *objective symptoms*.

The symptoms have a history. It may be the brief one of sudden onset, or a long one of rise and fall, of ebb and flow, of the mingling of complex phenomena from time to time. The story of the evolution of the disease is written as the *history of the present disease*.

The present illness may be due to or modified by previous disease. We may be consulted for the effects of one link in a chain of morbid disorders which began in infancy or early adult life. We should, therefore, learn of the occurrence of *previous disease*. Certain types of constitution and some few diseases are transmitted by parents to offspring; we should, therefore, inquire into the *family history*. A further insight into the nature of the suffering may be obtained by a knowledge of the age, sex, habits, occupations, environment, etc.; in short, by a knowledge of the *social history*, for, if the cause of the disease under consideration, or of conditions influencing the health value of the organism, is determined, a distinction from other affections with allied phenomena can frequently be made.

After the history of the patient has been taken objective symptoms are sought for by the use of the senses of sight, of touch, of hearing, with the instruments of precision to aid them—the *physical examination*, and by chemical and bacteriological methods.

The phenomena of disease are ascertained, therefore, by inquiry and by observation. The facts or data thus collected and the discriminate interpretation of them constitute diagnosis.

Methods of Diagnosis.—Inquiry.—The facts of the social history, the family history, the previous medical history, the history of the present disease, and the subjective symptoms are secured in this manner. At one time they were almost the only data that was collected. Their value is based upon experience and analogy and their interpretation is based upon deductive reasoning, a method not in vogue in scientific research. The truth or falsity of the facts gathered in this way cannot be proved by observation or research. As long as this method was employed, medicine could not be considered a science. A diagnosis based upon such data has been called an *empirical diagnosis*.

Observation.—The objective symptoms consist of signs evident of themselves, and of signs elicited by special methods of exploration. Signs not at once manifest, but elicited by special methods of examination are known as *physical signs*. They are restricted to phenomena secured by physical exploration, chiefly in the examination of the chest and abdomen, hence we have physical signs of disease of the chest and the abdomen. Modern methods of investigation have given to medical science instruments of research which, to secure accuracy, are employed in the laboratory. The use of instruments of precision, and of chemical and biological methods is rapidly bringing medicine within the domain of science. Certainly diagnosis, for these reasons, has made a decided advance. The number of diseases that can be positively diagnosed in the laboratory has markedly increased in the last few years.

Object of Diagnosis.—The object of diagnosis is to determine the condition of the living patient who may be suffering from disease. Diagnosis implies that the phenomena of disease are detected, *clinical diagnosis*; that the effects of the disease on the organism and the nature of the morbid process are ascertained, *pathological diagnosis*. In addition to naming the disease and its cause, we should include in the diagnosis a determination of the state of the disease and the recognition of its complications. Even this is too restricted an idea of diagnosis. It should include also the recognition of the cause of the morbid process; in other words, the *etiological diagnosis*. Moreover, diagnosis implies such a knowledge of the patient's condition as will enable the physician to estimate the possible dangers and the outcome of the disease—the *prognosis*.

As it is not disease that we treat, but a patient with an ailment, a full knowledge of the patient and his environment, mode of life habits, occupation, etc., should be obtained. The practical result of diagnosis is the ability to remove or prevent the occurrence of morbid processes, or to mitigate their effects by rational therapeutics.

Methods of Reasoning.—But we should not only secure facts, should also be able to utilize them for analysis and induction—

result of which is the formation of the diagnosis. The diagnosis is obtained by three methods—the *differential*, the *indirect*, and the *direct*. By the differential method the diagnosis of one of a few possible diseases is made after passing in review the positive and negative data. An indirect diagnosis is made by exclusion. Thus, a symptom group may suggest several diseases; each affection must be passed in review and excluded until one is found which closely corresponds to the data of the case under consideration. It is not one condition, because of the absence of certain symptoms; it is not another because of the presence of certain essentially different symptoms; and so a negative proposition is proved. In the direct method the data collected are sufficient to warrant a positive conclusion. The direct method is scientific, rational, and the most practical. It is a process of purely inductive reasoning.

Clinical Sense.—It is seen that observation and reasoning are essential in diagnosis. Something more is desirable, though not possessed by all observers. Long-continued precise observation develops in the student a *clinical sense*. He acquires the ability to place a precise value upon the symptom or sign, and to form with the various facts secured a clinical picture of the disease under consideration. The student will soon learn that under varying conditions the same symptom may have different values in different individuals. In consequence, the picture is true or false as the value is properly or improperly rated. So rapidly does the observer with this gift arrive at a conclusion that it is often said he is a diagnostician by intuition. If such a faculty were possible, it would be a valuable possession; but the ability to make a "snap" diagnosis is acquired only by patient, repeated observation, by systematic arrangement of the knowledge acquired and by fixing it on the mind.

Diagnosis Sometimes Impossible.—We are often unable to make a diagnosis. This arises when premises are wanting for the process of induction. The subjective symptoms may not tally with the known processes of disease; or the narrator of the history of the present disease may omit important evidence from lack of memory or knowledge, from design, or for other reasons. The objective phenomena may be developed in an ill-defined way; or they may be obscure, as the state of the abdominal contents in a person who is obese; or they may point to one or more processes the subjective symptoms of which are not present. At the time of observation the disease may not have developed fully. Under these circumstances a provisional diagnosis must be made and the final conclusion held in abeyance. If there is a suspicion that the patient is suffering from a contagious disease, the latter should for sanitary reasons be given the benefit of the doubt; and if the patient's condition requires prompt remedial action, the symptoms must be viewed first of all as possible indications for therapy.

Avoid Haste.—If prompt action is not required, haste should be avoided. It is not necessary to make a diagnosis at once, and it is

not a confession of ignorance if time is asked before an opinion is given. Repeated observation and reflection should be employed before a conclusion is arrived at. This applies particularly to those conditions which result from improper environment, for the proper detection of which social data, knowledge of temperament, etc., must be acquired. Then, again, it may be necessary to observe the patient under changed circumstances, or to study the effects of diet on gastric secretion or of the function of other organs. Haste leads to faulty diagnosis, and therefore to misdirected therapeutics.

Diagnosis Should Not Be Limited.—It is not sufficient to give a name to a group of symptoms and be satisfied that the diagnosis is made. The exact physical condition of the patient must be ascertained, and the functional powers of all the organs correctly determined. We thus learn if the more evident disease is the single expression of a malarial process, or if it is only a surface storm, the currents of which are underneath. A pleurisy or pneumonia may be the outcome or a complication of a latent nephritis. A peritonitis may be the sequel of an appendicitis or pyosalpinx, or diseases due to the same process may exist at the same time in two or more organs, as suppurative pleuritis and pericarditis. In such a case it would obviously not be sufficient to recognize one of the affections alone.

It is not sufficient, for instance, to recognize a neuralgia or a spasm. The state of the patient on account of which the neuralgia developed must be ascertained. Attention should be called to the importance of not being lulled into a false security by the belief that the diagnosis of the first day is sufficient. Complications may arise or the morbid process invade new territory. Thus, in the course of pneumonia, in a few days a meningitis may arise or an ulcerative endocarditis ensue.

Case Records.—Records of cases should be kept for obvious reasons. The habit compels a general survey of the case, and tends to prevent oversight in the examination. It naturally aids in the training of the powers of observation. It teaches precision in the narration of cases. The memory is aided by repetition and by lack of haste in ascertaining phenomena. The data are on record for more mature reflection, and to aid in the study of the literature of similar cases. The record is of value in case the patient returns for advice after a lapse of time. It may be of medicolegal value. The mental effect on the patient is good, for the taking of notes requires time and accuracy, studie observation. In case it is desired to study a large number of case records are scientific data. The records may be kept on loose sheet and filed for future use. When a sufficient number are secured, they may be classified and bound in volumes devoted to the various diseases. They may be noted in a blank book. At the end of the year the is indexed according to the diseases and the names of the patient; better method is the card system. The card-board should be eight inches. One or more cards devoted to each case are arranged catalogued according to the library system of card catalogues.

Method of Record.—A systematic plan should be pursued in noting the cases. It need not correspond to the lines of inquiry in the examination of the patient, which are modified by the circumstances of the case.

The following outline explains itself. The various data should be recorded in sequence, and in such manner that the facts of each line of investigation can be readily culled for review and analysis.

RECORD OF CASE No.

DIAGNOSIS	RESULT
Name.	
Residence.	
I. <i>Chief Complaint.</i>	
II. <i>Present Illness.</i> —Data, mode of onset, and probable exciting cause of present trouble; evolution of the disease to date of examination, with the subjective symptoms.	
III. <i>Social History.</i> —Age; sex; race; married or single; children, the number and health; miscarriages; place of birth; former residence.	
<i>Occupation:</i> Present and previous home surroundings, sanitary condition, etc.	
<i>Habits:</i> Tobacco; alcohol; tea; narcotics; sexual habits; regularity of meals; character of food and method of eating; number of hours of sleep; degree of fatigue; brain use; exercise.	
IV. <i>Family History.</i> —Hereditary tendency; health of parents, brothers, sisters, etc. Cause of death and age at which it occurred.	
V. <i>Previous Medical History.</i> —Previous illnesses; character of convalescence; syphilis and gonorrhea; injuries.	
VI. <i>Physical Examination.</i> —External appearance: development, color, figure, height and weight, attitude, expression of face.	
Temperature, perspiration, eruption, swelling. Condition of limbs and joints.	
<i>Examination of the digestive apparatus:</i> Mouth, tongue, gums, and pharynx; abdominal organs.	
<i>Examination of the respiratory apparatus:</i> Nose, mouth, and larynx; the lungs; inspection, palpation, percussion, auscultation, mensuration.	
<i>Examination of circulatory apparatus:</i> Inspection and palpation of cardiac area, percussion, auscultation of heart; similar examination of arteries and veins; the pulse; the blood-pressure.	
<i>Examination of the urinary apparatus:</i> Kidneys, ureters, and bladder.	
<i>Examination of the nervous system:</i> Intelligence, subjective nervous phenomena, sleep, gait, station, reflexes, paralysis, tremor, pain, convulsions, headache, disturbance of sensation, disturbances of speech, the organs of special sense.	
VII. <i>Laboratory Examination.</i> —Examination of blood, sputum, urine, other excretions, secretions, exudations, etc.	
Provisional diagnosis	
Prognosis	
Treatment	

CHAPTER II

THE HISTORY

THE history of a disease includes not only the present illness but also the past medical, family, and social history, so that facts are elicited which may have some bearing directly or indirectly upon the disease which may arise as a sequel of previous illness, as a result of inheritance, or from habits.

THE HISTORY OF THE PRESENT DISEASE

Scope of Inquiry.—The subjective symptoms of the disease are first elicited so that we can note the patient's intelligence, expression, and general bearing, and at the same time ascertain the direction further inquiry should take. The first step in the procedure is to secure in the patient's own words a knowledge of what troubles him most. This is known as the *chief complaint* and should be expressed in a few words. With this brief information in mind, immediate measures may be taken for the patient's relief if necessary, or, if not necessary, there have been suggested the most possible lines of search for the cause of the disease, and we can proceed to the history of the present illness, which includes an account of the *subjective symptoms*, of the *duration* of the disease, of its mode of *onset*, and of the *evolution* of the symptoms to the date of examination. These facts enable one to determine the events of the disease and the chronological order of their occurrence, that is, the clinical course of the disease. They are best learned in the language of the patient. If the memory fails or the symptoms are not clearly narrated, judicious questions may be put to complete the story; but leading questions must never be asked or only after the patient's own account has been fully given.

Mode of Onset and Duration of the Disease.—It is well to learn if the onset of the disease was *sudden* or *gradual*. If the former, the most striking phenomena are to be ascertained; a chill, convulsion, sudden vomiting, a profuse diarrhea, each points to lines of further inquiry. If the latter, did it follow upon an acute illness, or did each symptom gradually increase in intensity, and as each week or each month passed by, new phenomena creep into the symptom-complex? We thus learn if the affection under consideration is acute or chronic—its *duration*. It must not be forgotten that certain affections may be two or three days, or, on the other hand, as many weeks, in developing, which are nevertheless acute; typhoid fever is a good example. It is to

be remembered, also, that diseases may have sudden acute expressions, and that a chronic disease may be in existence a long time without the patient's knowledge. An acute diarrhea or a convulsion is often the first intimation of a chronic nephritis, and an attack of angina pectoris the first symptom of organic heart disease of long standing.

Evolution of the Disease.—In making inquiry concerning the evolution of the subjective symptoms, the *frequency, duration, character, and degree of severity* of each symptom, and its relationship to the function of the organ apparently affected, must be inquired into. Thus in the case of pain in the abdomen, we must learn its character, frequency, duration, intensity, and location, and whether it is associated with functional disturbance of any of the viscera in which the pain presumably has its origin.

THE SOCIAL HISTORY

The facts to be ascertained in the social history are the age, sex, occupation, habits, present and past residence, and opportunity for exposure to diseases.

Age.—Each period in evolution and involution of life has its peculiar physiological processes susceptible to variations by external influences. In infancy there arise congenital affections, accident incident to parturition and to improper management of the cord, disturbances of digestion and of the pulmonary tract.

In childhood, one is prone to suffer particularly from the exanthemata, cerebrospinal meningitis, chorea, anterior poliomyelitis, disorders of the lymphoid structures, bronchitis, bronchopneumonia, endocarditis, whooping cough, diphtheria, mumps, bone tuberculosis, gastro-enteritis, and intussusception. At puberty the perversions and sequels of earlier conditions are liable to occur, increasing in frequency as adolescence advances. Anemia, chlorosis, exophthalmic goitre, hysteria, acute tuberculosis, and the acute infectious diseases are particularly likely to occur at this time of life. *In the middle period* of life, diseases that arise from occupations, from habits and from exposure, and from child-bearing are seen, as lead-poisoning, hepatic cirrhosis, lobar pneumonia, and gall-stones. Furthermore, the diseases, the predisposition of which is inherited, as gout, diabetes, and cancer, angina pectoris, aneurysm, arteriosclerosis, myocarditis, nephritis, and other diseases largely due to degenerative vascular changes make their appearance, in many in the latter part of midlife, in others they do not appear until old age arrives.

Sex.—The diseases, characteristic of the respective sexes, arise because of a difference in anatomical structures and physiological functions and because of the difference in exposure to varying causal agencies. Diseases more common in the male sex occur on account of occupation, from exposure, from overactivity of mind and body, and finally from the

formation of bad habits. The diseases that are more prevalent in the female sex, apart from affections arising out of menstruation and child-bearing, find their origin in the more or less sedentary nature of women's lives, and, hence, the opportunity for introspection; hysteria, neurasthenia, and the neuroses abound in them. For inexplicable reasons males are more subject to epilepsy, syringomyelia, locomotor ataxia, hemophilia, gout, diabetes, Addison's disease, leukemia, appendicitis, chronic endocarditis, and cancer of stomach or rectum. Females are more subject to exophthalmic goitre, gastric ulcer, gall-stones, chorea, and cancer of liver and bile passage as well as of the genitalia and breast.

Occupation.—Each occupation demands effort in one particular direction or compels exposure to deleterious influences. Writer's cramp, emphysema, eye-strain, laryngeal affections, and a series of disorders arise from excessive use of the affected parts. Miners, stonecutters, grinders, and those employed in similar occupations are subject to chronic bronchitis and emphysema, which in turn invite tuberculous infection. Lead-poisoning, mercurial poisoning, phosphorus-poisoning, arsenical and brass-poisoning are incident to the occupations which compel contact with these poisons. Infections from animals are to be thought of in tanners, hostlers, and others coming in contact with animals and their products.

The manner and degree of employment of the mind should be inquired into in functional and organic nervous disorders.

It is not to be forgotten that the occupation at different periods of life must be found out, the age at which life's economic battle began, and the circumstances that surrounded the early career. The deleterious influence of a former occupation may be appreciated only after the patient is in an entirely different sphere of labor.

Habits.—Habits as to clothing, as to hours of rest and sleep, as to character of food, time, regularity, and manner of eating (the indigestions, gout), as to exercise, as to the use of alcoholic stimulants (cirrhosis of the liver, neuritis, brain affections), tobacco (amblyopia, cardiac palpitation), of tea or coffee, of narcotics, must be inquired into. Methods of work, methods of recreation, domestic joys or sorrows, must be ascertained. A knowledge of the habits, of the life, of the marital relations, of the individual, is essential to a rational diagnosis, and hence a true therapeutics.

Present and Past Place of Residence.—Town residence and country residence, a residence in a damp locality, by the sea and in the mountains, in particular valleys, in different water-sheds, in tropical or frigid clime, each makes an impress on the constitution even if actual disease is not created. Regions infested by the various forms of animal life which act as hosts in the transmission of disease (malaria, yellow fever, plague, Malta fever, echinococcus disease, etc.), goitre districts, localities in which vesical calculi are common, or in which special epidemic diseases abound, as yellow fever, cholera, or dysentery, are matter for inquiry.

The sanitary arrangements, drainage, ventilation, water-supply, and heating of the dwelling are to be scrutinized.

Exposure to Infection.—The probability of exposure to a contagious or infectious disease must be looked into and the presence of epidemics ascertained. The period of incubation and the prodromal symptoms must be known.

THE FAMILY HISTORY

The family history is elicited to determine the affections which may or may not be hereditary. Strictly speaking a disease is rarely inherited unless congenital, but the tendency or predisposition to it is inherited just as the same facial characteristics or mental traits of a family are found in one generation after another.

To secure accurate data, the age and state of health of parents, brothers and sisters, if living, are ascertained; or if dead, the cause of death and age at which it took place. Similar questions may be applied to several generations of the family and to collateral branches.

Difficulties.—A correct family history may not be secured because of family pride, because the patient may not understand the medical terms, or because the inquirer forgets the interdependence of many diseases, or because the patient uses obscure terms ("dropsy," "teething," "cold," "change of life," etc.), or symptoms ("jaundice," "chills and fever," "diarrhea," etc.).

Obscure Terms.—Care must be exercised to secure definite data, and not to overestimate statements. Thus, when the cause of death is stated to be dropsy, or jaundice, or a cold, or teething, or change of life the term is meaningless. Control questions must be put to determine the character of the symptoms that attended the fatal illness.

Common Morbid Processes.—The data of the family history are of no avail unless it is remembered that many fundamentally identical affections have various modes of expression. Various diseases may be allied to the one suspected to exist in the patient, and be overlooked because of this difference of expression. One member of a family may die of heart disease, another of rheumatic fever; or some may have had chorea, or cutaneous affections, or renal calculi; such ailments are possibly expressions of the same morbid process. Finlayson well puts them into groups and fittingly portrays them as follows:

"In regard to tuberculous diseases, we ask for swollen glands or waxy kernels, or running in the neck, diseases of the spine and other bones, bad joints, white swellings, disease of the glands, of the bowels, water in the head, consumption of the lungs, or decline, or weakness of the chest, with spitting of blood and so on.

"Heart disease, rheumatism, chorea, psoriasis, and some other cutaneous affections, and perhaps renal concretions and emphysematous bronchitis, appear to replace each other in different members of the same family. The neurotic group includes the various forms of neuralgia, epilepsy, hypochondriasis, hysteria, and insanity.

"Apoplexy and hemiplegia in their hereditary character are associated with vascular disorders. Gout, disease of the liver, contracted kidney, renal calculus and gravel, and angina pectoris form another allied group; and these have also some affinity with the disorders connected with arterial degenerations. Syphilis, which, of course, has marked hereditary characters, assumes such a multitude of forms as to preclude enumeration; but the tendency is for such syphilitic diseases to fail in the course of time from early death or sterility. Abortions, stillbirths, early deaths in infancy, associated with cutaneous eruptions on the buttocks and with snuffles, are important in many family histories; nervous deafness, opacities of the cornea, notched teeth, epilepsy, and imbecility are occasional manifestations of the same disorder in those children who survive."

Inherited Diseases.—(1) *Hemophilia* is the most striking affection that is transmitted by inheritance. (2) *Syphilis* may be inherited. No investigation is complete in certain eye, skin, nasal, or throat affections, in diseases of the nervous system, and to a less degree in other ailments, without settling the question of ancestral syphilis. (3) A tendency to the diseases or perhaps the metabolic causes productive of them is inherited in *emphysema*, *arteriosclerosis*, *diabetes*, *tuberculosis*, *cancer*, *gout*, *rheumatism*, and *many skin affections*. It is well to remember that members of families subject to these diseases, particularly arteriosclerosis, are usually short-lived and age prematurely and that many families seem to possess great powers of resistance to infectious diseases, and are as well not subject to inherited diseases so that their members are usually long-lived. Furthermore, certain families have a predisposition to many minor ailments and diseases especially gastro-intestinal affections.

Nervous Diseases.—Nervous diseases are the most common of inherited diseases, as progressive muscular atrophy, hereditary chorea, Thomsen's disease, Friedreich's ataxia, migraine, asthma, epilepsy and forms of insanity. To these may be added family types occurring generation after generation, or racial characteristics, manifested in hysteria, hypochondriasis, and other psychoses or neuroses. By *neurotic heredity* we mean that in certain families a tendency to the development of various forms of nervous disease exists, which may be manifested, however, only in certain members of a generation. The indications of neurotic heredity are manifold. Inquiries must be made in regard to insanity and epilepsy, to instances of suicide, to peculiarities of character, to criminal tendencies, to addiction to the use of drugs such as alcohol or opium; to congenital deformities; or to congenital diseases, such as deaf-mutism. Certain forms of nervous disease, the causes of which are unknown, are spoken of as hereditary or familiar because two or more examples have been observed in the same family.

Terminology.—Various terms are employed to indicate the nature of an inheritance. *Direct inheritance* means that the child acquires the disease before or at birth. If both parents have the disease the child

THE HISTORY

is likely to suffer more severely, and this is spoken of as *cumula inheritance*. By *indirect inheritance* is meant the condition in the collateral ancestry and not the parents have had the disease. parents may appear to be healthy, although the grandparent earlier ancestors, in the direct line have suffered from the same disease and this is called *atavistic inheritance* (atavism, the skipping of a generation). By *similar inheritance* is meant the occurrence in the offspring of a disease similar to but not identical with that from which the parents have suffered. Examples of such diseases are Huntingdon's chorea and Goldflam's periodic paralysis. By *dissimilar inheritance* is meant the development in the offspring of a form of disease differing from that which existed in the parents, as an epileptic child born of parents suffering from neurasthenia, hysteria, or insanity.

Contagious Diseases.—In the inquiry it may be well to ascertain the probability of disease being transmitted from husband to wife, or the opposite. Syphilis, gonorrhea, and tuberculosis are examples. No doubt may this probability apply to the transmission of disease from husband to wife, but to their offspring as well.

THE PREVIOUS MEDICAL HISTORY

The medical history is secured to determine (1) if the present disease has occurred previously, (2) if it is the sequel of a former disease, or if a similar affection has occurred, and may therefore be excluded from the diagnosis.

1. The following diseases are likely to recur: asthma, bronchitis, delirium tremens, erysipelas, gout, gall-stones, malarial fever, meningitis, tonsillitis, rheumatism, renal colic, diphtheria, apoplexy, angina pectoris, intermittent hemoglobinuria.

2. The diseases which have sequelae are syphilis, gonorrhea, scarlet fever, rheumatic fever, tonsillitis, and other general infectious diseases. Gall-stones, renal calculi, local infections, as of the appendix or bladder, are often followed by secondary disorders. Nephritis, myocarditis, myocardiitis, aneurism, angina pectoris, apoplexy, heart disease, pancreatitis, intestinal obstruction, locomotor ataxia, leukemia, amyloid disease, fatty degeneration, pelvic inflammatory disease, and the various manifestations of syphilis are disorders which most frequently, secondarily to other diseases. Injuries are often the cause of tuberculous bone disease, epilepsy, pachymeningitis, meningitis.

3. The diseases which are likely to occur only once are most exanthemata and typhoid fever. In the diagnosis of obscure diseases if a history of their occurrence is ascertained, they can usually be excluded. The data of occurrence and character of the disease, its duration, the degree of severity, and the completeness of convalescence must be determined.

SECTION II

SUBJECTIVE DIAGNOSIS, DATA OBTAINED BY INQUIRY

CHAPTER III

GENERAL SUBJECTIVE SYMPTOMS

The subjective symptoms are expressive of the sensation of the patient, and vary in accordance with the sensibilities of the individual affected. Thus acute pain may apparently represent a severe process in one, while in another the same severity of process may be represented by the minimum amount of pain.

The paresthesias—that is, the abnormal, disagreeable, or perverted sensations the patient feels are not diagnostic of any particular affection when diffused over the whole body or referable to more than one organ, but are evidence of ill-health or malnutrition. When, however, they occur in a definitely localized area or part of the body, they are usually indicative of disease, the result of irritation of the sensory nerves supplying the part. The fallacy of depending upon these symptoms for a diagnosis, as indeed of all subjective symptoms, is that they may be feigned, imagined, or exaggerated.

1. *Numbness or tingling and burning* may be general or local. Numbness, when local, may precede the eruption of herpes zoster, may be the aura of an epileptic attack, the premonitory symptom of neuritis, or a symptom of neuritis. It may be of reflex origin, from irritation at a distant point; or it may be toxic, as in carbolic acid or acetate poisoning. The sensation is complained of in neurasthenia and hysteria, and is a common accompaniment of locomotor ataxia, multiple neuritis, and chronic spinal meningitis.

2. *Flushing*, and a constant sensation of heat, with or without perspiration, attend the percolation of the menopause, and are common in uterine disorders, in chronic gastritis, and neuritic conditions generally.

3. *Numbness*, or a sensation of numbness with a symmetrical tendency, occurs in acute, suppurative disease, pulmonary thrombosis, and pneumonia. This symptom is also present when the action of the circulation is interfered with, likewise venous stasis, pain, and shock may cause it. It occurs in neurasthenia, and frequently accompanies pathological acts like abortion.

GENERAL SUBJECTIVE SYMPTOMS

4. *Formication*, or itching, is a variety of pruritus attending skin affections, hysteria and neurasthenia, many organic spinal and brain tumors, toxic conditions, as lead-poisoning, uremia, gonorrhea, jaundice, and is at times premonitory of cerebral apoplexy. Some drugs (ergot and copaiba) give rise to it. It is a local symptom affecting the genitals, in diabetes or when there is leucorrhea, and the anal seat-worms or hemorrhoids.

5. *Throbbing* may be felt over the entire body in aortic regurgitation, cardiac hypertrophy, hysteria, neurasthenia, and anemia. Pulsation of the aorta may be functional or due to aneurism. Throbbing head and neck is seen in exophthalmic goitre. It is a local sensation limited to the aorta or the heart in aneurism, in palpitation, and in cardiac hypertrophy.

6. A sense of *fulness* located in the chest or epigastrium is seen in chronic gastritis, dilated stomach, hypertrophy of the heart, emphysema, and asthma; or to distention of the abdomen, as from tympanites.

7. *Oppression* or *weight* in the chest is found in cardiac and pulmonary affections and in hysteria; in the epigastrium, in gastric neurosis; in the head, in neurasthenia and hypochondriasis or as an *aura*; in the abdomen or pelvis, when a tumor is present.

8. *Girdle sensation* occurs in spinal cord disease, or temporarily after violent cough or vomiting, and in diaphragmatic pleurisy.

9. *Precordial distress* is found in angina pectoris, in cardiac weakness and dilatation, in acute forms of indigestion and diarrhea, and in hysteria and neurasthenia.

10. *Chill and Fever*.—Both are subjective as well as objective phenomena, but as one can be accurately estimated by an instrument of precision (thermometer), and as both are generally associated with objective signs, their discussion will be postponed. (See Objective Signs.) An abnormal sensation of cold or of heat will be discussed in the chapter on Nervous Diseases.

11. *Coldness* may be due to anemia and to myxedema, as well as to functional nervous disorders. When localized, with pain or paresthesia, it may be due to one of the forms of mild neuritis. It may occur in locomotor ataxia or syringomyelia. (See Sensation in Nervous Diseases.) It occurs locally in conditions which interfere with the circulation to the parts affected, as arteriosclerosis, phlebitis, and thrombosis of the vessels of the extremities.

12. A feeling of *strength*, or the idea of an ability to perform feats of strength or endurance, or a great mental feat, is a subjective symptom that is dwelt upon by the patient who is developing or passing through certain stages of parietic dementia. It is accompanied by other evidences of exhilaration. *Exhilaration* attends chlorosis and some forms of hysteria and neurasthenia, the physical or mental exhaustion of strength taking place in the after part of the day and even upon undue excitement. Corresponding depression usually follows.

GENERAL DIAGNOSIS

of weakness, exhaustion, or fatigue is often complained of when an absolute demand is made upon the bodily strength, it can otherwise it is not exerted. The patient complains of being tired in the morning than upon retiring, or of a sense of weariness in performing accustomed or special duties. Mental depression attends the phenomenon. It is due to neurasthenia generally, a frequent accompaniment of the forms of toxemia to which rheumatism belong; of the toxemia of certain varieties of tobacco, alcohol, and other narcotic poisons (tea or mineral poisons). The same sense of fatigue attends the early stage of the specific fevers. It is a symptom that has been frequently of late in the sequential period of influenza. A sense of weakness must not be confounded with true weakness or prostration, seen in diabetes, tuberculosis, exophthalmic goiter, and anemia. While the patient is aware of its presence, it is not considered as under the objective phenomena of disease, for it is not a recognized sign of disease.

A sense of pressure or heaviness is usually the result of pelvic disease, constipation, or an overfilled bladder.

A sense of tightness or constriction is felt in the first or dry stage

In the following chapters the subjective symptoms which are common to many diseases, but suggestive of a certain disease, are considered. These symptoms cannot be considered as symptomatic of any one disease or as indicative of a fundamental causal condition should be searched for. A sense of heaviness or of indicating a pulmonary disorder may be due

CHAPTER IV

PAIN

Definition.—Pain may be defined as a sensation known to everyone which produces on the part of the organism, as a whole, the desire to abolish or escape from it.

Pathology.—The pathology of pain is generally believed to be a state of impaired nutrition, and hence of injury, gross or microscopic, either at the periphery or in the afferent nerve tract. There is also the so-called sympathetic or reflex pain, due to irritation in a part removed from the locality to which the sensation is referred.

Pains in reference to the general nervous system may be classified according to the localization of the lesion into (1) peripheral, (2) central, and (3) general. Peripheral pains are local and due to some alteration either in the structure or in the nutrition of the peripheral nerves. The disturbances may be situated at the sensory terminations, or anywhere in the course of the nerve or nerve roots. Pains due to causes situated in the latter place are usually perceived at the peripheral distribution of the nerve, and are, therefore, spoken of as referred pains. The nature of central pain is not at present clearly understood. General pains are those due to some toxic condition of the blood or impairment of the nutrition of the nervous system as a whole, and manifested as pain in the regions of least resistance.

Variations in Disease.—Pain is, perhaps, the most variable symptom in disease. It ranges from a sensation of mere discomfort, as the dull ache of chronic lumbago, to the stabbing pain of pleurisy or the intolerable anguish of heart pang. It is at times compatible with the highest mental endeavor or the severest physical exertion, at other times the whole energy of the organism is absorbed in resisting it. It may be definitely localized in any part of the body, in any of the tissues, or distributed over an ill-defined area.

The Recognition of Pain.—The Mode of Expression.—As a rule the physician learns of the existence of pain by the complaints of the patient. Thus he learns more or less accurately its location, character, degree, and duration, and usually something of its causation. There are also other ways by which suffering is expressed which may be grouped among the objective symptoms. They are:

Facial Expression.—The expression of the face is the most common interpreter of the emotions, and is far more reliable than the patient's statements.

Pulse Rate.—Pain causes an increase in the pulse rate, in general the more severe the pain the more rapid the pulse, a sign of pain that is absent in malingering and hysteria.

Attitude.—Not less characteristic are the various postures as the sudden fixing of heart pang; the retracted head of meningitis; the immobile side of pleurisy; the crouching attitude or restlessness of colic; the flexed thighs and immobile trunk of peritonitis; the shuffling gait to the affected side in renal colic; the bent knee of articular rheumatism.

Reflex Actions.—Further, there are certain reflex actions that are associated with local irritations; thus the closing of the eye on irritation of the conjunctiva; or the limp, characteristic of pain moving or resting the weight of the body on an affected limb. There is the sudden shrinking of the whole body; the attempts to defend the part, or the sudden movement of the hand to the affected part, or the sudden jerking away of the part itself if the act be possible; these are reflexes, and sufficiently diagnostic of local suffering.

Sources of Error.—Unfortunately, pain is one of the most unreliable of symptoms. It is necessarily a subjective symptom, with innumerable qualitative as well as quantitative variations. The particular degree in either respect is of importance in diagnosis, and only the roughest means, if any, are available to estimate it objectively; the physician is compelled to rely almost wholly upon the statement and appearance of the patient.

Errors as to the presence or absence of the degree of pain arise because the patient may exaggerate his sufferings or belittle them. The tendency to exaggeration is most marked in certain persons: nervous temperament, in those suffering from chronic disease of long standing, in those accustomed to indoor and mental labor, in women and in the young. The tendency to depreciation is most marked in the phlegmatic temperament, in those accustomed to hardship, especially if of small intellectual development, in men, in the aged, and those under strong mental inhibition from religious or other excitement.

Simulated Pain.—Simulated pain is to be recognized by the existence of a motive for deception.

Malingering.—Simulation is common in those who seek damage for injuries, or in those who have a morbid craving for sympathy and attention. Its detection depends upon the skill of the physician, who by distracting the attention from the part complained of, observe that the pain disappears, or, on the other hand, that pain is admitted in a part to which attention is directed; moreover, the physician observe an absence of adequate physical alteration, and usually inconsistency in the symptoms.

Hysteria.—The so-called hysterical mask is of much value, for the bitter complaints and the placid or even smiling features cannot fail to strike the observer by their incongruity. True hysteria is apt to be deceptive. The difficulty is increased because actual physical changes occur, as amaurosis with dilatation of the pupil, contracture, and induration about the joints, unquestionable anesthetics and palsies. True hysteria is often to be detected only after prolonged and painstaking study of the case.

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the stomach or intestines, or any of the hollow viscera; (6) in angina pectoris, the crises of locomotor ataxia, and in acute brow ache or trigeminal neuralgia.

The onset may be *gradual*, and followed by continuous increase in intensity or variation. Such onset indicates that the process is one of slow development. It is observed in various forms of rheumatism, in inflammation of muscles and of mucous membranes, in chronic inflammations of serous structures, in chronic bone disease, and in slowly developing mechanical pressure, as by tumors.

2. *DURATION*.—The duration of pain also indicates the acuteness of chronicity of the causal morbid process. Pain of *short* duration is seen in the affections in which it develops suddenly (see Mode of Onset), in acute serous inflammations, and in neuralgias. Pain of *long* duration, if constant, is usually due to organic lesions; if intermittent, it may be due to neuralgia.

Pain is also divided, as to duration, into temporary and constant pain. *Temporary* pain indicates total or partial abeyance of the morbid process, while *constant* pain points to its continuance.

Pain may also be intermittent or remittent, paroxysmal or periodic: (a) *Intermittent* and *remittent* pains are characteristic of neuralgias, or point to a functional origin; they recur because the cause which superinduces them again becomes operative. (b) *Paroxysmal* pain is the form which occurs when there is obstruction of channels, as the bile ducts in biliary colic, the intestines, the uterus, and the ureters in the various forms of colic to which they are liable. The paroxysms of pain recur in the course of the attacks. (c) The term *periodic* is applied to pains that occur at distinct intervals. The toxic headaches and nerve headaches, as migraine, are often periodic.

3. *TIME OF OCCURRENCE*.—*Diurnal* pains are usually reflex from functional disorders. Some pains, as headaches due to cardiac weakness and to forms of anemia, are present during the day, because the patient is in the upright position; they disappear in the recumbent position, and hence are absent at night. Neurasthenic pains are worse in the early part of the day. *Nocturnal* pains usually due to periosteal inflammation are common in syphilis.

The *time-relation* of pain to functional acts is of importance. Thus in gastric pain its relation to the taking of food is to be ascertained. Pain coming on before meals is gastralgia, due to hyperacidity. Occurring after meals, it is due to ulcer, cancer, or gastritis, sometimes to gastric neuritis. Occurring several hours after, it may be due to pyloric obstruction or duodenal ulcer. Chest pain, increased by the act of breathing, is muscular or pleuritic.

4. *CHARACTER*.—The character suggests the location and the cause of pain. *Sharp, burning, gnawing, or stabbing* pain is usually due to inflammation of serous membranes, to various forms of colic, and to forms of neuralgia. *Throbbing* pain is a sharp form that occurs in throbbing colic. *Cramp* is pain due to sudden spasms of muscles or to

spasm of the intestines, as in colic. *Gnawing* or *boring* pain attends the pressure necrosis of aneurism, and is characteristic of gastralgia or hyperacidity. *Throbbing* pain is usually associated with acute inflammation, whether superficial or deep. It may be rhythmic with the pulsations of the heart. *Dull* pain is due to slow, chronic inflammation in the bones and the viscera; it is the pain of myalgia and of fatigue in the muscles. It may be of an aching character. But *aching* pains may also be general; they are found among the prodromata of the acute diseases, attend and follow a chill, and occur in most characteristic forms in influenza and dengue. *Pressing* pain is complained of when it attends an attempt to remove material from the viscera as the passage of water when the bladder is inflamed; the passage of feces in dysentery. The term *tenesmus* is applied to it, so that we have vesical tenesmus and rectal tenesmus. The passage of clots or other material from the uterus is attended by pain with pressure or "bearing down," as it is termed.

Finally, the character of pain is often an indication of the nature of the disease as well as of the tissue affected. (1) Thus boring and constant pain is seen in bone and periosteal disease. (2) Soreness or aching in muscular affections. (3) The pain is sharp and stabbing when serous membranes are affected. (4) Smarting and burning, or, perhaps, dull pain and soreness, when mucous membranes are inflamed. (5) Burning or itching in affections of the skin. (6) Dull and usually constant in visceral affections, although in malignant disease of various organs it may be sharp and paroxysmal. (7) Aching, burning, and throbbing in a nerve trunk and its distribution, with tenderness, commonly indicate neuritis. (8) A sense of swelling, distention, or bursting attends the pain of obstruction, as in renal or hepatic colic. (9) Rending or tearing pain may be complained of when a hollow viscus or sac is ruptured, as notably in rupture of the sac of extra-uterine pregnancy.

5. LOCATION OF PAIN.—Pains may be general or local.

A. *General Pains*.—In syphilis, malaria, lead-poisoning, in the early stages of most acute infectious diseases, and toxemias generally there are general pain, soreness, and fatigue. General muscular soreness is a constant accompaniment of rachitis and scurvy. General pains are not confined to the muscles, but may be seated also in the fibrous structures or bones. In their more severe forms such pains occur in dengue, which is also known as "break-bone fever."

B. *Local Pains*.—The location is, in general, an indication of the seat of the disease. It may be accepted as an almost universal rule that pain due to a local process is limited to the immediate or associated nerve supply of the diseased region.

Referred Pains.—The above statement holds true even when the referred pains—that is, those felt in the associated nerve supply—are as far distant from the site of the morbid process as the knee pain of coxitis, the shoulder pain of hepatic disease, pain in the neck from

pericarditis or diaphragmatic pleurisy, the ear and temporal pain of lingual carcinoma, the pain in the legs from cancer or ulcer of the rectum, the testicular and thigh pain of renal colic, or the umbilical pain of vertebral disease.

Local pains may be (a) superficial or deep-seated; (b) circumscribed or diffused; (c) bilateral; (d) unilateral.

(a) *Superficial* pains are due to involvement of the superficial nerves distributed to the skin or to the muscles directly underneath, or to the structures in close relation to the skin, as the peritoneum, the pleura, or the pericardium. *Deep-seated* pains, when in the extremities, are due to bone disease; when in the abdomen, to disease of the viscera, particularly inflammatory affections, to aneurism, or to vertebral disease; when in the chest to disease of the aorta or mediastinum.

(b) *Circumscribed* pain is always due to a limited area of disease, or is reflex. *Diffused* pain indicates involvement of a large area with less intensity of process than when the pain is circumscribed. When the pain is diffused, or as it is sometimes called, radiating, over an area of nerve distribution, its point of origin may be found somewhere in the course of the nerve, either in the trunk or in one of its branches, as the foot pain of sciatica.

(c) *Bilateral, symmetrical, and superficial* pains indicate a central or bilateral cause; while, on the other hand, (d) *unilateral* pain implies that the causal lesion is one-sided.

C. *Peripheral Pain of Central Origin*.—In meningitis and other general organic affections of the brain and cord, peripheral pains are frequent, and may be the earliest and most striking symptoms. For example the pains in the joints, of central origin, may be constant, or paroxysmal and lancinating when the disease is chronic. (See Character.) The paroxysms of pain may be most excruciating and sometimes cause collapse. They are known as *painful crises*. Pain may be complained of in various viscera, as well as in the joints. Sudden, intense pain, without functional disturbances of the affected viscera, occurs independently of any lesion of the part or of any apparent exciting cause. One class of these attacks has received the name *gastric crises*. The pain is in the epigastrium, and is associated with vomiting. In another class *laryngeal crises* occur, with pain in the larynx and violent spasmodic cough with dyspnea, the pain extends over the shoulders. Pains in crises also occur in the muscles. Crises occur chiefly, if not entirely, in locomotor ataxia. They are characterized by their sudden onset, their extreme severity, the absence of organic disease at local cause in the affected viscera, the sudden termination, and the normal condition between the attacks.

6. *Pain Modified by Pressure, Movement, Heat, or Mental Processes*.—We also study pain under the influence of pressure, movement, temperature, rest, etc. Pain may be abated or excited and exaggerated by pressure. *Gastralgia* and *colicky* pains in the intestine, which may be nervous in origin, are relieved by pressure.

particularly if the whole hand is applied. Pain from the dislocation of an organ, as a movable kidney or displaced uterus, or from dependent viscera, may be relieved by judicious pressure in the proper direction, so as to correct the displacement. The excitation or exaggeration of pain by pressure is called *tenderness*. Pain from affections of the nerve trunks can be distinctly localized by pressure in the course of the nerve trunk, and particularly at the points where the cutaneous filaments of the nerves come through the fascia. These tender points in the thorax are along the vertebral column, in the axillary region, and anteriorly about the sternal line—the points of Valleix. We distinguish neuralgias from myalgia by the presence of these tender points. Tenderness usually denotes a superficial inflammatory process, though deep-seated inflammation also results in the production of the condition. It is a sign of particular value in the diagnosis of intra-abdominal inflammatory lesions. Thus appendicitis, salpingitis, hepatic abscess, cholecystitis, etc., all have as a cardinal symptom tenderness on deep pressure over the involved part. Occasionally pain is elicited in visceral disease, by superficial pressure on the skin of the area supplied by the nerves from the spinal segment enervating the viscus. This *cutaneous hyperalgesia* may extend also to the muscles causing muscular contraction, rigidity. An example of this is seen in cases of renal colic where the rectus muscle of the side affected often becomes distinctly rigid. At the same time tenderness may be found over the vertebræ at the level of that portion of the cord from which the nerves which supply the areas of hyperalgesia have their origin. Tenderness is sometimes present over the first to fourth thoracic vertebræ in cardiac disease; over the fourth to eighth vertebræ in stomach diseases; the eighth to twelfth vertebræ in hepatic affections, and over the lower lumbar and upper sacral vertebræ in diseases of the rectum or genitalia.

Pain increased by *movement* points to an affection of the bone, muscle, joint, or nerve in the part moved. Some few pains are relieved by movement of the body only because the mind is diverted in this act. Pain, when superficial and increased by movement, is due to neuritis, myalgia, or rheumatism.

Almost all pains are modified by *rest*. Its influence has but little diagnostic significance.

Pain modified by *temperature* (cold or heat applied to the spine, ice, or hot water in a sponge) and by *electricity* usually gives information as to the seat of the disease in the spinal column, of which the pain is the external expression.

Pain modified by *climate* is rheumatic or neuralgic; if modified by weather or season, it is due to neuralgia or neuritis. Pain is often modified and mollified by fixing the attention of the patient on some other part or on some extraneous subject, when the previously alleged tender area may be pressed upon without causing any evidence of suffering. The admission of the occurrence of such pain, with other

evidences of hysteria, points to the underlying causal factor in the production of pain.

Varieties of Local Pain.—Pain in the Head.—Pains in the head may be classified, according to location, into those due to affections of the superficial structure, those due to affections of the cranium, and those due to intracranial conditions.

1. **SUPERFICIAL STRUCTURES.**—These are to be further classified as those of the skin, those of the muscles, and those of the nerves.

The Skin.—A feel of tension, with soreness, accompanies the dermatitis of erysipelas. Intense local irritations are caused by burns and scalds. Hyperalgias of the scalp frequently accompany meningeal and cranial affections.

The Muscles.—Sharp pains in the occipital or frontal region, increased by wrinkling the scalp or brief pressure, but generally relieved by firm and constant pressure, and occurring with irregular periodicity, are suggestive of myalgia or fibrositis. This latter condition (fibrositis) is frequently associated with headache—the so-called indurative headache—which is often induced by changes in the weather. In the latter stages small areas of induration may be palpated in the affected muscles.

The Nerves.—Neuralgia occurs in paroxysms located in the course of the nerve trunks and presenting points of sensitiveness, while the nerve enlarges from the skull and where it divides for cutaneous distribution. The character of the pain is variable; it may be of the most acute or rending form, or, but more rarely, a persistent dull ache; it may be throbbing or occur in successive paroxysms at brief intervals, or it may be regularly periodic. The local neuralgias of the supra-orbital, dental, auricular, and occipital nerves are the most common seats of this pain. In neuralgia of the trigeminus or *tic douloureux* the entire fifth nerve or one of the three main divisions is affected. The pain is often extremely severe and associated with trophic changes in the areas supplied by the affected nerve. In the great majority of cases the pain is unilateral.

Pain simulating neuralgia is frequently due to some local irritation; foreign bodies have been known to cause paroxysmal attacks for a number of years until removed; diseases of the bones are a prolific source, especially in the case of the jaws and the cervical vertebræ. Enlarged cervical glands occasionally irritate the great auricular and small occipital nerves. Bilateral occipital pain is very characteristic of cancer of the cervical vertebræ. In these cases there is usually pain on movement of the head or pressure upon it, and some stiffness of the neck. Intracranial growths occasionally cause pains, usually paroxysmal, limited to one of the branches of the trigeminus.

Reflex Neuralgia.—Certain of the cephalic nerve pains are symptomatic of disturbance in the associated but distant nervous distribution. Thus, neuralgia of the supra-orbital nerve is supplied by the ophthalmic division of the fifth

will produce ocular and supra-orbital pain. Errors of refraction of the eye cause the same kind of pains. Pain in the temporal region and in the external auditory meatus is often due to intense irritation of some of the branches of the inferior dental nerve; the usual cause is cancer of the tongue, but irritable lingual ulcer may also produce it, and even severe inflammatory conditions of the lower jaw. Pain may be caused in the ear alone when there is irritation of the inferior dental nerve.

Systemic Neuralgia.—Perhaps in the majority of cases of cephalic neuralgias the cause is to be found in some systemic disturbance, as gout, diabetes, malaria, anemia, syphilis, and crises of locomotor ataxia.

Pain in Special Parts.—Localized dull burning pains, accompanied by febrile symptoms, indicate inflammations of the mucous membranes of the nose. Severe nasal pain is the result of foreign bodies in the nostrils. A dull persistent headache located just beneath the eyebrows often accompanies coryza, and indicates extension to the frontal sinuses. It is sometimes intense and agonizing.

Inflammations of the conjunctiva produce local pain, usually causing the sensation of a rough foreign body. Iritis and inflammations of the deeper structures of the eye cause pain in the eyeball and supra-orbital pain. The pain of glaucoma may simulate trigeminal neuralgia.

Most ulcers of the mouth are comparatively painless, though simple and tuberculous ulcers are very irritable.

In inflammatory or ulcerative lesions of the pharynx and larynx the pain is usually sharp and cutting and exaggerated by swallowing and talking.

Pain at the angle of the jaw increased on swallowing, almost invariably unilateral and associated with swelling of the parotid, is unmistakably due to parotitis.

The neuralgias and inflammations of the middle ear are exceedingly painful; they may consist of a sharp continuous pain, or a series of regular exacerbations and remissions, or a throbbing sensation; pain often radiates to the jaws and side of the face.

2. *AFFECTIONS OF THE CRANIUM.*—A dull, constant headache, limited to a small area, later increasing in severity, and the pain assuming, perhaps, a boring character, tenderness, often very severe, over the affected area, and probably slight edema of the scalp, with some rigidity of the muscles of the neck, and the ordinary signs of the inflammatory process indicate inflammation of the cranial bones.

3. *INTRACRANIAL HEADACHES.*—Intracranial headaches are functional or organic. Both forms may be acute or chronic. The typical acute functional headache is seen in the more or less common type known as migraine or hemicrania.

Migraine is a periodic sensory neurosis characterized by pain in the distribution of the trigeminus and other cranial nerves. The headache is usually unilateral, and, as it is probably due to vasomotor

disturbances, is always associated with vasomotor symptoms. It is often hereditary.

The pain is most frequently felt on the left side of the head first. It may be seated in the anterior frontal, temporal, or parietal region. The pain is continuous, and increases in intensity to the height of a paroxysm. The painful points are not usually detected.

Chronic Headaches.—Chronic functional headaches are usually habitual in the sense that the attacks are constant, but there may be longer or shorter intervals of freedom from pain. The pain may be confined to one part of the head, but usually is not limited to the distribution of a particular nerve.

Causes.—(1) Hemie: anemia. (2) Toxic: (a) lead and other mineral poisons, alcohol, the toxins of uremia, tobacco; (b) diathetic states (gout, diabetes); (c) infections (malaria, syphilis, specific fevers). (3) Neuropathic states (epilepsy, neurasthenia, chorea, hysteria, neuritis, fatigue). (4) Reflex causes (ocular, nasopharyngeal, auditory, gastric). (5) Condition associated with heightened blood pressure. (6) Organic disease.

Headaches are divided according to their situation into frontal, occipital, parietal, vertical, diffuse, and combinations of any of these forms. The most common forms are the frontal, the fronto-occipital, and the diffuse. The situation of the headache is frequently difficult to determine and may occur anywhere in the head irrespective of the lesions. In the following conditions the usual site (at least of the origin) of the headache is mentioned. Ocular headaches are usually frontal when due to errors of refraction. When due to muscular insufficiency they are occipital and cervical. Nasopharyngeal headaches are dull, and frontal or diffuse. When the pharyngeal tonsil is enlarged the headache may be dull, frequently recurring, and seated in the occipital region. In follicular tonsillitis and in obstruction of the Eustachian tubes the headache is diffuse. In disease of the middle ear it is temporal and occipital. Gastric or dyspeptic headaches without constipation are often occipital, sometimes frontal. With constipation and intestinal irritation they are diffuse and frontal. Neurasthenic headaches are usually associated with a sense of pressure or weight, and are seated in the frontal and vertical regions. In spinal irritation the pain is of a boring character and situated in the occipital region. The earliest symptom of the neurasthenic headache is neck weariness and pain in the neck. Headaches occur in brain workers when the brain and eyes are overtaxed. Headaches in epilepsy are severe, and are limited to the vertical or occipital regions.

Organic headaches are usually violent, associated with fulness and throbbing of the head. They may be remittent, becoming more intense with each exacerbation. Organic headaches may be due to inflammation, to abscess and softening, to tumor, to congestion of the brain, or to inflammations of the meninges. Anything that increases the quantity of blood in the vessels of the head will increase the pain.

particularly in organic headaches. In acute inflammation of the brain the pain is agonizing, continuous, associated with vomiting and fever, and sometimes delirium. In abscess of the brain the pain is less violent. It is occasionally paroxysmal and attended by paralysis and disturbed cerebration. In tumor of the brain the headache is severe and paroxysmal. In congestion the pain is dull, increased in stooping, and by bodily or mental fatigue. Some congestive headaches are due to violent exercise, and are relieved by bleeding at the nose. In all congestive headaches the face is flushed, the bloodvessels are turgid, and the vessels in the eye-grounds are overfilled. In meningitis the pain is constant, is more or less fixed, and sometimes very sharp. Syphilitic headache is frontal or temporal, worse at night, and often periodic. Inflammation of the accessory sinuses causes severe persistent pain over the involved sinus.

Headaches are divided according to the character of the pain: (1) Pulsating and throbbing. (2) Dull and heavy. (3) With constriction, squeezing, or pressing. (4) Hot and burning. (5) Sharp and boring. The headaches of the first class are often associated with vasomotor disturbances, as in migraine. To the second class belong the toxic and dyspeptic headaches; to the third, the neurotic and neurasthenic; to the fourth, the anemic; to the fifth, the hysterical, neurotic, and epileptic. Vertigo is a common accompaniment of the dyspeptic type of headache situated in the frontal regions. Somnolence is more marked in the syphilitic, anemic, and malarial headaches. Nausea is more common in occipital forms of headache.

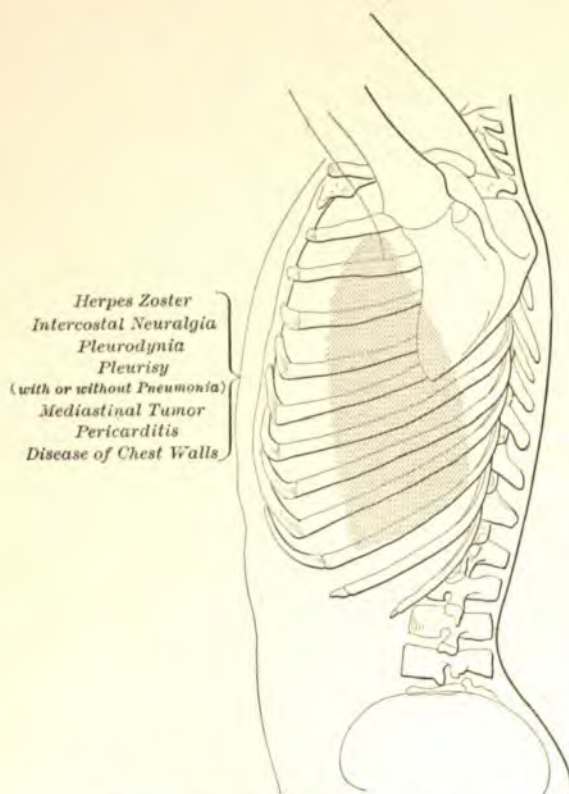
Duration.—Eye-strain causes occipital pain, which is rarely persistent, but comes on after prolonged use of the eyes. It may be associated with headaches in other parts of the head, due to other causes. In chronic meningitis the headache is persistent and located in the vertex or parietal regions. When thickening of the meninges, with adhesions, takes place from trauma, there results constant pain, with frequent exacerbations and sensitiveness of the head. Uremic headache is not constant but persistent headache may be present in the later stages of Bright's diseases and in diabetes. In cerebral and general arteriosclerosis or other conditions of hyperpiesis, pain in a part or the whole of the head is common; it may be persistent, though subject to exacerbations in case of excitement or violent exercise. Headache following study, in children, is due to brain strain, to eye-strain, or to indigestion. Persistent headache is sometimes due to asthma. In rare instances headache is said to be idiopathic. Neuralgic headaches are usually periodic, and may be associated with throbbing or pulsations. They are associated with vasomotor signs. Hysterical headaches are irregular and shifting; they persist after all causes are removed; they are replaced by pain in other parts of the body. They are usually associated with other manifestations of hysteria.

Pain in the Thorax.—Pains of reflex origin are seated usually in the shoulder or the back and are due to liver or gastric disease. The pain

of liver disease is referred to the right shoulder; of ulcer of the stomach, to the interscapular and lumbar regions, or to the top of the shoulder. Pain is rarely a symptom of disease of the lungs unless the pleura is involved.

In *bronchitis* there may be some soreness and some oppression behind the sternum, but otherwise pain is absent.

FIG. 1



The site in general of thoracic pain in various conditions.

In *pleurisy* pain occurs as a result of the fibrinous exudation. It is sharp and lancinating, exaggerated by breathing, and so severe as to impede the respiration and to cause the cough to be short and catchy. It is seated usually at the base of the chest, in the lateral or anterior region. The pain of pleurisy may be increased by local pressure, but is always relieved by general pressure which limits respiratory motion. In the pleurisy that attends phthisis pain is quite common, being of the same character as the pain of acute fibrinous pleurisy, but varying in situation and in degree. It occurs in paroxysms and follows a slight exposure to cold,

undue exertion, or fatigue. It may persist for twenty-four hours, and then disappear, until brought on again by a repetition of the cause. It must be distinguished from the myalgia of phthisis, which is due to cough and exposure.

In *myalgia* the muscles and fasciæ at the body attachments are very tender.

The pain of pleurisy must be distinguished from pleurodynia, from intercostal neuralgia, and from the pain due to disease of the ribs.

In *pleurodynia* the muscles are sensitive if palpated or compressed between the fingers. A large area is affected, but the physical signs of pleurisy or of pneumonia cannot be elicited. Usually there is a history of recurrent myalgia.

Intercostal neuralgia is sometimes difficult to distinguish. The pain is sharp, localized, and may modify the movements of the chest. General pressure relieved it; local pressure at the points where the terminal filaments of the nerve come to the surface may increase it. The so-called Valleix's tender points, however, are not always present in cases of intercostal neuralgia. Cough and physical signs are absent.

Pain in the lateral thoracic wall is an accompaniment of herpes zoster, also of distention of the stomach and colon.

Fracture of the rib or *caries of the rib* may be recognized by the physical signs of these conditions, and by the local tenderness. Both may, however, be attended by localized pleurisy, indicated by more severe pain on coughing or on deep breathing.

A neglected *empyema* that is about to point will cause pain in some area of the chest. The pain is seated usually at the points of election for the discharge of the empyema, as the fifth interspace on the left side.

More or less constant pain at the apices, when undoubtedly independent of affection of the muscles, is a questionable sign of *tuberculous disease*. It may be aggravated by pressure.

Pain behind the sternum is often a reflex neurosis from gastric disorder. It may occur in bronchitis. It may be due to cancer of the mediastinum, to aneurism, to aortitis or angina. Pain in the sternum or ribs is syphilitic, or is due to periostitis or necrosis, for example, following typhoid fever, rarely to cancer. Chronic fibrous inflammation of one or more of the attachments of the muscles is of common occurrence. The pain lasts for years; it is persistent, sometimes associated with stiffness; it is increased by movement, and there may be extreme aching pains in the parts.

Pain in the lateral wall of the thorax may also be due to a dilated stomach, a loaded colon, or to inflammatory disease of the liver or spleen, as well as the conditions just mentioned. Pain in the breasts may be due to croupous pneumonia, or in women to disease of the breast, or reflex from pelvic conditions. Pain may be present at the xiphoid in diaphragmatic pleurisy.

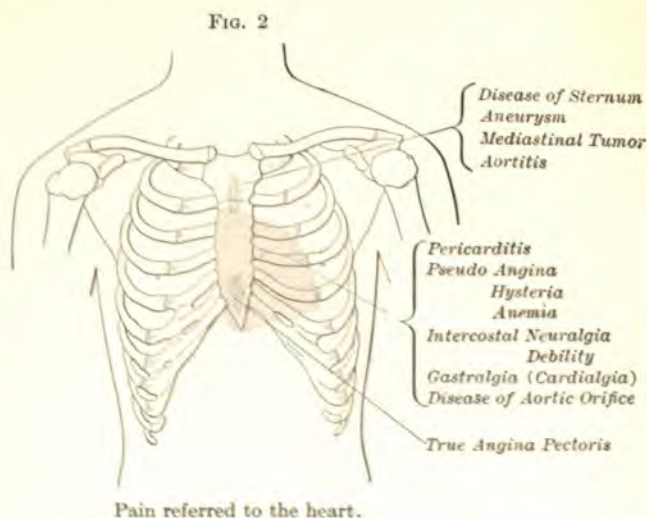
Pain Referred to the Heart.—1. IN DISEASES OUTSIDE OF THE HEART.—Pain in this region is usually due to causes other than disease of the

heart or of the pericardium. It may be located in the precordia, or more precisely in the fifth or sixth interspace on the left side and in the epigastrium.

The causes are (1) neuralgia, (2) pleurodynia, (3) myalgia, (4) local pleurisy, (5) periostitis, (6) aneurism, (7) abscess.

The *neuralgias* may be associated with points of tenderness. The tenderness corresponds with the position at which the nerves have their exits through the fascia to the surface, and are found along the sternum, in the midaxilla, and along the vertebræ. The callus of an old and forgotten fracture of the rib may pinch the nerve.

In *pleurodynia*, probably an affection of the pleural nerves or of the muscles or their attachments, the pain is more diffuse, is increased by pressure with the finger tips, relieved by pressure with the whole hand.



In *myalgia*, which is seen so frequently in phthisis on account of severe coughing, in rheumatism, and in debilitated subjects generally, the pain is more or less diffuse, interferes with the movements of the chest, is relieved by uniform general pressure, and is associated usually with myalgia in other parts.

The pain of *pleurisy* is recognized because it usually inhibits the act of breathing and is associated with cough; friction sounds may be detected.

Periostitis.—The pain is associated with tenderness and swelling. One or more of the costosternal articulations may be extremely tender from the periostitis of syphilis, or from that which follows typhoid fever.

The pain may be due to internal pressure and erosion of the ribs from *aneurism*. This affection may cause neuralgic pains.

Abscess.—Pain may be due to a localized tuberculous abscess between the pericardium and the walls of the thorax, or to empyema.

The Epigastrium.—Pain in the epigastrium is often held to be due to cardiac disease, but as a rule it is of gastric or gall-duct origin. The pain of angina pectoris may, however, be limited to this region. Acute, severe, and excruciating pain in the epigastrium may be due to rupture of the heart, and also to pericarditis.

2. THE PERICARDIUM.—Pericarditis is a common cause of pain in the region of the heart. The pain may radiate to the left shoulder and extend down the arm. It is paroxysmal and may have some of the characteristics of angina pectoris. It is increased by movement, by pressure, and by the action of the diaphragm. It may be referred to the epigastrium. The patient is often obliged to sit up in bed, and suffers from orthopnea. A pericardial friction sound is usually detected.

3. THE AORTA.—In syphilitic aortitis the pain may extend along the course of the aorta, or it may be referred to the sternum. The pain is usually dull, and continuous, often with paroxysmal exacerbations, especially angina pectoris. There is usually valvular disease at the aortic orifice. With ordinary atheroma there may also be more or less continuous dull substernal pain often associated with anginal attacks.

Aneurism.—The pain usually results from pressure of the aneurism upon adjacent structures. The pain is of a boring character, localized at one point, if the aneurism presses on a bone and causes erosion. The pain is of a dull, aching character, increased by movement, relieved by rest or by change of position; or it may be acute and of a neuralgic nature when nerves are pressed upon. It may follow the course of the nerves and be associated with numbness and tingling. The long duration of the pain, its localization, and its aching character are sufficient to exclude angina pectoris. When the pain is unilateral, it may be due to pressure of an aneurism upon the nerves at their exits from the vertebral canal; the pain extends along the course of the intercostal nerves. It is severe and burning, but there are no localized points of greater intensity. The pain may extend down the arms, and when the abdominal aorta is affected, it may extend down the legs. If rupture of the aneurism takes place, the pain is sudden and sharp, and death ensues quickly.

4. THE HEART.—Three forms are seen: (1) Pain due to disturbances of the rhythm. (2) Pain due to valvular disease. (3) Pain due to angina pectoris.

Pain Due to Disturbance of the Rhythm.—In the large majority of cases, palpitation and irregularity of the heart occur without pain. Paroxysms of palpitation are sometimes attended with severe precordial pain and distress, as in reflex palpitation; in palpitation of Graves' disease and of anemia. The palpitation of organic disease is induced by exertion. The rapid action of the heart is painful and the throbbing causes distress.

Pain Due to Valvular Disease.—Pain is of more frequent occurrence in disease of the aortic valves. Constant dull pain and pseudo-anginoid attacks are of frequent occurrence in mitral stenosis.

Pain Due to Angina Pectoris.—The pain of angina pectoris is severe and agonizing. It comes on suddenly, lasts but a brief interval, and recurs in paroxysms. The patient realizes that the pain is in the heart, and complains of feeling as if the organ were held in a vise. There is a sense of impending death, with sinking and oppression. From the heart the pain radiates to the neck and down the arms. It extends particularly to the left arm, and may be severe in the wrist or at the tips of the fingers. Prostration follows the attack usually, but the precordial distress disappears entirely.

The points upon which the diagnosis is based are:

1. The Seat of the Pain: This is usually behind the middle or the lower part of the sternum, and more to the left than to the right, or in the epigastrium. Thence it extends to the posterior portion of the axilla or it may radiate to the neck. In some instances it extends to the occiput. Frequently the pain extends to the left arm as far as the elbow or even the fingers. It may extend to the abdomen or to the right arm. I have seen it affect both arms. It is not influenced by external pressure.

2. Character: The sense of constriction with the indescribable torture is most characteristic.

3. Respiration: The respirations are shallow, or may even cease, but there is no dyspnea.

4. The Position of the Patient: The attitude is one of fixation.

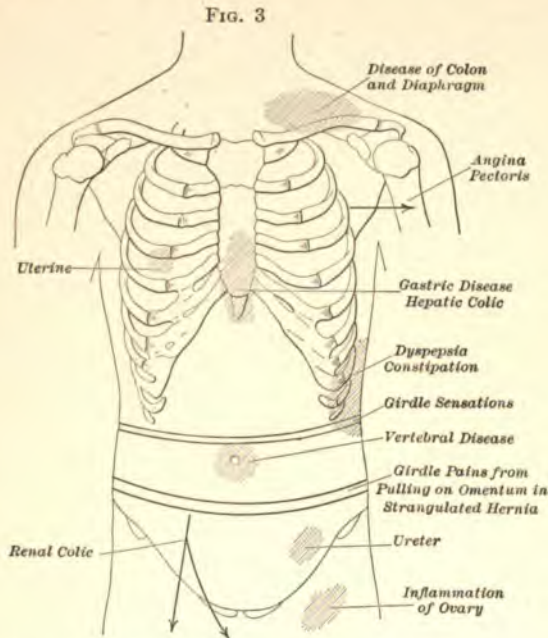
5. The Expression: The pale face, extremely anxious countenance, and the cold sweat on the forehead, make a striking picture, which when once seen can never be forgotten.

6. The Pulse: During the attack the frequency of the pulse is not much influenced, and the action of the heart may be uniform and regular. Scarcely its frequency may be lessened. The tension of the pulse is increased during the attack.

Spasmodic pseudo-angina or false angina from neurotic, vasomotor, or toxic causes can be distinguished only with difficulty. It occurs much more frequently than true angina. The attacks most frequently come on at night and may be periodic. The pain is less severe and the oppression is not so marked in pseudo-angina; coldness of the hands and feet with the occurrence of syncope, or a general feeling of sinking, are common symptoms. The pain is of long duration and is associated with great agitation. It is preceded by neuralgia, and neuralgic pains persist after the attack. It is often relieved by antineuralgic medication.

Pain of the Abdomen.—*Pain Referred to the Abdominal Walls.*—The skin, the nerves, the muscles, and fascia, the connective tissue, may be the seat of pain, or the pain may be referred. A common cause of abdominal pain is disease of the vertebra, with pressure upon the

peripheral nerves at their emergence from the spinal column. The pain is situated in the median line, either below the ensiform cartilage or around the navel. It is an intermittent pain. Aneurism of the abdominal aorta, causing erosion of the vertebræ by pressure, gives rise to the same kind of pain.



Referred pain in various disorders.

Pain within the Abdomen.—Pain in the abdomen may be sudden in onset, or it may represent the culmination of slight sensations of discomfort progressively increasing in severity. Sudden acute pain points to inflammation, perforation of one of the hollow viscera, gastralgia or enteralgia, flatulent distention of the stomach or intestines, or to obstruction of one of the numerous channels. Thus, in a case of gastric ulcer, sudden pain suggests that perforation may have taken place; chronic pain indicates the existence of some chronic process such as ulcer, or a gastric or intestinal neurosis. Sudden paroxysmal pain is spoken of as *colic*; the successive spasms being often attended with vomiting, rapid pulse, coldness of the extremities, cold sweat, and more or less collapse, except in lead colic. Such pain is seen in intestinal, hepatic, renal, and in uterine or vesical colic.

Pain due to obstruction is of a wave-like character radiating from a point beyond the site of the obstruction.

When the pain is *general* it points to myalgia, to intestinal colic, to peritonitis, to tympanitic distention, or under certain conditions

as discussed in the following pages to hepatic or renal colic and other conditions.

Local pain, for example over the liver or spleen, is generally due to involvement of the peritoneal coverings of these organs, and partakes of the character of local peritonitis. It may, however, be due to extreme congestion, to malignant disease, or to displacements.

PAIN IN THE EPIGASTRIUM.—Pain in this situation (excluding the abdominal walls) may be due to: (1) Localized peritonitis or perigastritis, which may be secondary to infection or injury of the peritoneum from disease of contiguous organs. (2) Affections of the pancreas. (3) Aneurisms of the aorta or of the celiac axis. (4) Disease of the gall-bladder and gall-ducts. (5) Disease of the vertebræ. (6) Diaphragmatic pain from pleurisy or violent coughing or in pneumonia. (7) Appendicitis. (8) Impaction or ulceration of transverse colon. (9) Affections of the stomach, gastric pain.

Pain of Gastric Origin.—In diseases of the *stomach* pain is a very common symptom, and presents all degrees of severity, from a mere sense of discomfort or uneasiness to agony. In mild gastritis of whatever origin, there may be only a feeling of weight and fulness, or uneasiness or discomfort after eating, often relieved by belching. *Cardialgia* is a form of discomfort in the epigastrium scarcely amounting to pain, but attended by heartburn or acidity. The pain of gastric crises in locomotor ataxia is gnawing and cramp-like, frequently attended by partial collapse.

In *ulcer* there is more or less constant feeling of soreness in the epigastrium, while after taking food the dull pain is aggravated, and becomes sharply localized. Frequently there is pain in the back at the same level a little to the left of the spine and between the mid-scapular region and the lumbar vertebræ. The pain usually occurs sooner after taking food than in the case of cancer, and is more frequently relieved by vomiting. The pain may radiate to the arm.

When perforation of an ulcer occurs, the pain is sudden and severe and attended by collapse.

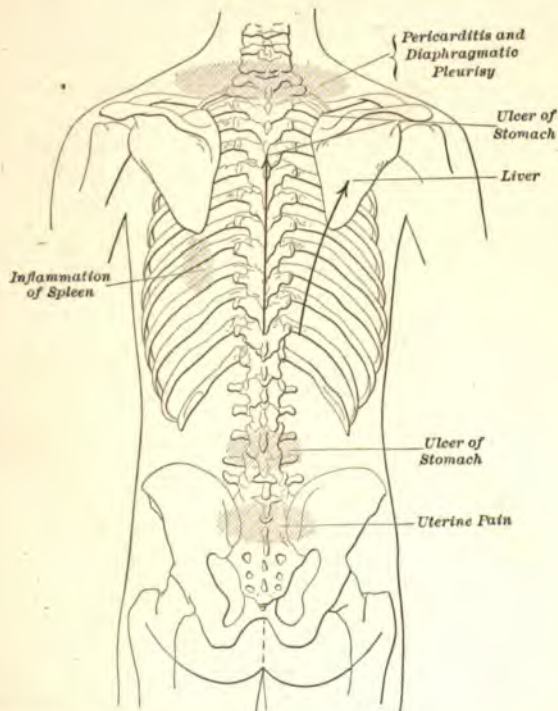
In *hyperchlorhydria* the pain may be exactly like that of ulcer, and it may be impossible to differentiate the two conditions. Usually, however, the ingestion of food relieves the pain of hyperchlorhydria.

In gastric *cancer* pain may be wholly absent throughout the entire course of the disease; but, as a rule, pain is more continuous than in ulcer, less severe, not so sharply localized, does not come on so soon after taking food, and is not relieved to the same degree by vomiting.

In *acute gastritis* the pain and its character vary with the intensity of the inflammation. When due to the irritation of some toxic agent that has been swallowed, the pain is severe and burning; when the result of imprudence in eating or drinking, the pain is of a dull, sickening character. In either case there is more or less tenderness on pressure. Sometimes, in mild cases of catarrhal gastritis, firm pressure from a broad surface affords at least temporary relief from the distress.

Time of Occurrence.—The significance of pain in gastric disease depends on the time of its occurrence. Pain coming on before eating or when the stomach is empty is usually due to hyperchlorhydria, though it may be associated with no alteration in the gastric secretions, and is then termed simply gastralgia; it is relieved by food. When it comes on after eating it is usually due to organic disease of the stomach, as ulcer or carcinoma; but it may be due to neurasthenia. When the pain is diffuse, it is due to hyperacidity and gas formation, as in dilatation, catarrhal gastritis and simple indigestion. When localized, it is due to ulcer or cancer, and is associated with tenderness.

FIG. 4



Direction of radiation of pain in various disorders.

Pain of Pancreatic Origin.—In disease of the pancreas, either from the passage of a calculi (rare) or from hemorrhage into the parenchyma, there may be sudden, severe paroxysmal pain. It may be severe in the back and extend up the thorax.

PAIN IN UMBILICAL REGION.—This is usually referred pain and may be due to (1) appendicitis; (2) perforation of a viscus; (3) hernia, umbilical; (4) disease of vertebræ; (5) gall-stones; (6) cancer of

omentum; (7) local or general peritonitis; (8) floating kidney; (9) gastropptosis; (10) intestinal pain.

Pain of Intestinal Origin.—Intestinal pain as a result of disease of the intestine is usually colicky in nature. The colic of intestinal indigestion occurs suddenly, or it may be preceded by other signs of intestinal indigestion. The pain is relieved by moderate pressure and by warmth. The pain at its height is described as agonizing, and of a boring or shooting character, abating after a time and then increasing again; it may shoot from the region of greatest intensity to the shoulders, back, chest, or iliac region. Prostration or collapse rapidly ensues. The pain may be relieved by the passing of flatus.

If the colic is due to *indigestible food*, it may have been preceded by an attack of acute indigestion, and the griping pains may have developed at long intervals, with gastric and intestinal flatulence. If the colic is due to *gas* alone, there is much tympanites; if it is due to *feces*, there is an antecedent history of constipation, and fecal masses may be detected in the rectum or in the course of the colon; if due to *plumbism* there is obstinate constipation. The presence of fever is against intestinal colic, and points to inflammation in some portion of the abdomen.

Intestinal colic must be distinguished from enteralgia. The latter comes on slowly and lasts for hours or days. The pain is situated around the umbilicus, and is relieved by deep pressure, although the skin may be hyperesthetic. Sometimes the abdomen is retracted; there are no signs of indigestion, and flatulence and borborygmi are absent.

Intestinal Obstruction.—Intestinal colic must not be confounded with organic disease of the bowels, with resulting obstruction. In these affections occur sudden constipation and rapid prostration. In *intussusception* the stools are characteristic. *Ileus* or *strangulation* is associated with a history of previous peritonitis or the presence of hernia; in the latter there may be characteristic signs at the hernial points. The pain may be transverse and with a sense of constriction if the omentum is incarcerated or adherent and drags on the mesentery. In obstruction from *external pressure* the presence of tumors have been known previously or can be recognized. In *fecal* obstruction or in obstruction by gall-stones the local signs may be pronounced, and the pain is usually in the ileocecal region. The affection is acute.

Girdle Pain.—This is a peculiar pain or sensation in the trunk, due to disease of the spinal cord. It is described as the sensation of a band drawn tightly around the body. It varies from a simple drawing sensation to extreme pain encircling the trunk. It is situated above the level of the umbilicus. In mild forms it is due to chronic myelitis or spinal sclerosis; in severe forms to inflammation of the nerve roots, or to cancerous, syphilitic, or tuberculous disease of the meninges.

PAIN IN THE RIGHT HYPOCHONDRIUM.—This may be due to (1) movable kidney or calculus; (2) subphrenic abscess; (3) pleurisy and

pneumonia; (4) pancreatic disease; (5) ulcer or cancer of stomach or duodenum; (6) appendicitis; (7) cancer, impactions or ulcerations of hepatic flexure of colon; intestinal obstruction; (8) localized peritonitis; and most usual (9) diseases of liver and bile passages.

Pain of Hepatic Origin or of Bile Passages.—Pain is a frequent symptom of liver disease. When sudden in onset, acute, and increased by pressure or movement, it is due to perihepatitis. Stabbing or darting pains belong to cancer. Abscess may be attended by pain from associated perihepatitis. A neuralgia or hepatalgia is at times undoubtedly present.

In hepatic colic the pain is situated in the region of the gall-bladder, and may radiate to the right shoulder or to the back. It is sometimes fixed in the right sternal line at about the level of the cartilages of the sixth and seventh ribs. The attack is attended by vomiting usually of bile-stained fluid; and may be followed by jaundice.

Pain in the right hypochondrium and epigastrium is probably caused more frequently by congestion of the liver than by any other cause. The congestion is secondary to cardiac disease as a rule, so that the cause of the pain is readily diagnosed.

PAIN IN THE LEFT HYPOCHONDRUM.—This may be due to most of the conditions that cause pain in the opposite side, but the most frequent cause of pain here is a perisplenitis, colitis, gastric conditions associated with flatulence, or movable kidney.

PAIN IN THE LUMBAR AND ILIAC REGIONS.—Pain in these localities may be due to (1) intussusception (right side) and volvulus (left side); (2) ulceration and inflammation of the cecum or sigmoid; (3) hernia; (4) typhoid fever; (5) pyelitis, hydronephrosis, movable kidney, perinephritic abscess, etc., and ureteral calculus; (6) inflammatory and other conditions of pelvic origin; (7) appendicitis; (8) movable cecum.

Pain in the Kidneys.—Pain of the kidneys is referred to the loins. Bilateral pain is complained of as a dull, aching pain, sometimes increased by movement, often attended by a sense of weight or pressure. Pain of this character extends over the entire lumbar region, and is due to disease of both kidneys, as renal hyperemia, nephritis, and bilateral pyelitis. It is generally mistaken for pain due to other causes, as myalgia or disease of the vertebræ. If myalgic, it may follow exposure to cold and be associated with pain in other muscles.

We have also unilateral pain. The pain may be seated in the region of the kidney behind, opposite the two lower dorsal and two upper lumbar vertebral spines, or deep in the abdomen, to the right or left of the spinal column, below the level of the umbilicus.

Unilateral pain may be constant or paroxysmal. Constant pain is usually due to organic disease of the kidney, as carcinoma or tuberculosis. (See Palpation.) It may, however, be due to the impaction of a calculus in the pelvis of the kidney. It may also be due to a displaced or movable kidney. In tumors the pain may follow the course of the sciatic nerve and simulate sciatica. In pyelitis and

hydronephrosis the pain is of a tearing character, whereas in movable kidney it is variable.

Paroxysmal and lancinating pain, the paroxysms occurring at intervals are usually due to the passage of a renal calculus, to obstruction or twist of the ureter, as in Dietl's crisis, or rarely to blood in the pelvis of the kidney. In renal colic pain begins in the kidney and thence extends along the ureter. It is always localized more to the right or left of the median line in the abdomen. It is more frequently in the lower portion of either of the upper quadrants, three inches to either side of the median line, depending upon the kidney affected. From this region the point of maximum intensity and of local tenderness moves in the lower quadrant in an oblique direction toward the median line, rarely an inch below the transverse umbilical line. The pain then extends to the region above the pubis and down the thighs. It partakes of the character of neuralgia in the intervals between paroxysms. It must not be forgotten that sometimes in case of disease of one kidney the pain is referred to the normal kidney.

Pain of Appendiceal Origin.—In acute appendicitis the patient is seized with sudden pain, which may be described as occurring in the lower right quadrant, but is sometimes complained of about the umbilicus. It frequently follows indiscretions of diet, and may be attended by vomiting; it is usually relieved by eructation, but not by the passage of gas, a point of great importance in the diagnosis. The attack lasts from twelve to twenty-four hours. It may be so severe as to cause collapse. In chronic appendicitis the pain may be dull or aching in character, often with acute exacerbations of sharp colicky pain. In the chronic as well as in the acute form there is usually tenderness of McBurney's point.

PAIN IN THE HYPOGASTRIUM.—The following conditions causing pain are the most frequent ones found in this region: (1) cystitis; (2) cystic calculus; (3) distended bladder; (4) other diseases of the bladder; (5) inflammatory or other disease of the pelvic organs; (6) dysmenorrhea; (7) ectopic gestation.

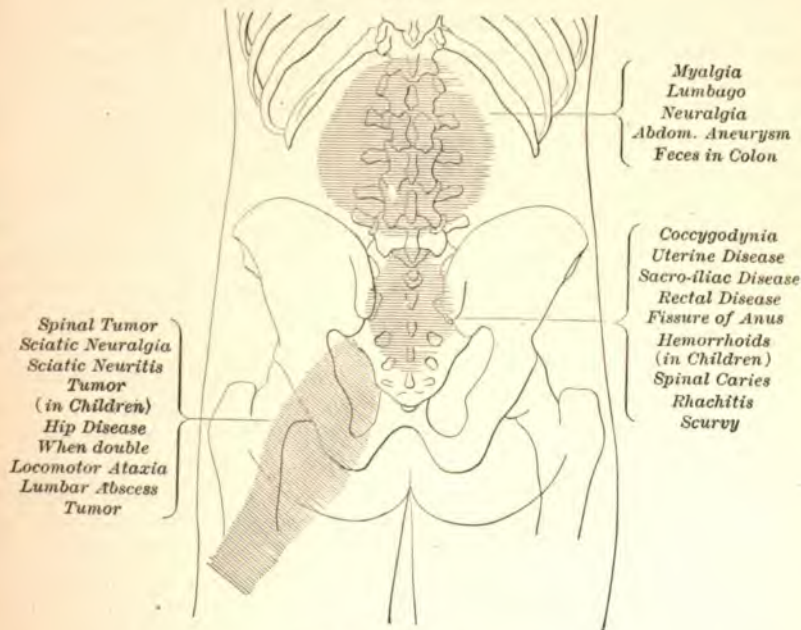
Pain in the Back.—**PAIN IN THE SPINE.**—Pain in the spine is due less frequently to organic diseases of the cord than to acute or chronic inflammation of the meninges, to disease of the bones of the vertebral column, or to curvature of various forms from muscle weakness. Rachialgia and tenderness in the course of the spine occur after concussion.

1. *Disease of the Spinal Cord.*—In organic disease of the cord pain may be referred to the loins, the sacrum, or to the parts about the spine, but not to the spinal column itself. We may have also the eccentric or radiating pains, of which mention was previously made, due to irritation of posterior nerve roots. These pains may be dull, resembling those of rheumatism. In acute cases the pains are accompanied by febrile symptoms which may suggest rheumatism, especially when the other spinal symptoms are in abeyance. In chronic cases

these peripheral spinal pains are influenced by the weather, and this likewise makes it difficult to distinguish them from the pains of rheumatism. Rheumatic pains in the limbs occurring after middle life, with or without joint changes, should suggest locomotor ataxia. In this affection sharp and darting pains, "pain crises," and girdle sensations occur.

2. *Disease of the Vertebrae*.—Fixed localized pain at some point in the vertebral column points to traumatic, syphilitic, or tuberculous caries, to arthritis deformans, or to pressure necrosis as by an aneurism. Pain

FIG. 5



Pain in the back.

due to vertebral disease is both local and radiating. It is increased by pressure directly on the spinal column (on the head), by heat or by cold, or by electricity applied over the part. It is relieved by removing the pressure of the weight above, as by raising the head or shoulders. It is relieved by the absolutely recumbent posture. With this pain the movements (flexibility) of the spine are interfered with because of spasm of the muscles or ankylosis; there may be deformity. When the patient is placed upon a flat surface the normal lumbar arch is changed. Tenderness over the vertebrae from visceral disease has already been discussed.

3. *Disease of the Meninges*.—Pain due to meningeal disease is local and radiating. It is associated with muscular spasm and rigidity of the spinal column.

4. *Spinal Curvature*.—The pains of curvature from muscular weakness extends along the nerves. The patient is afebrile. The signs of organic disease above mentioned are absent, but muscle weakness and general signs of debility are present. Pain in the spine frequently attends scurvy and rachitis. It may be accompanied by paresis of the muscles and closely resembles an organic brain or cord disease.

PAIN IN THE LUMBAR AND SACRAL REGIONS.—*Acute Pain*.—When acute, without fever, pain in the loins may be due to lumbago, to a sudden uterine retroversion, to a suddenly moved kidney, or to calculus of the kidney; with fever, acute Bright's disease, smallpox, muscular rheumatism, tonsillitis, influenza, dengue, or spinal meningitis must be looked for.

Chronic Pain in the Lower Back.—*Backache*.—Backache may be due to many causes. When the pain is in the region of the kidneys, the latter may be at fault (congestion-inflammations). Organic diseases may be associated with backache. More frequently pain, if in one kidney, is due to a calculus or to accumulations of uric acid gravel. Pressure over the kidney or a sudden jar from a false step will usually excite the pain. It may be constant in movable kidney. When low down, just above or over the sacrum, it is due to disturbance of the pelvic viscera. The uterus, the colon, and rectum (impaction, cancer) must be examined. Sacral pain is also due to disease of the sacro-iliac joints when the characteristic attitude and gait are present.

Otherwise we may have: (a) Pain due to affections of the muscles. (1) *So-called rheumatic myalgia*: Increased by movement, by dampness, by pressure often relieved by warmth, by the recumbent posture, or by rest. When the fasciæ or the ligaments of the vertebræ are affected, the upright position and pressure in small areas increase the pain; other muscles may be affected alternately. (2) *Myalgia from sprain*: A history of injury is obtained. Usually one side is larger than the other. Tenderness is present and movement increases the pain. There may be increased swelling, vasomotor disturbance, or ecchymoses. A neurosis of the so-called spinal or traumatic type (hysteria) attends the pain. (3) *Myalgia from fatigue*: Not only acute fatigue after exertion, but also chronic muscle-tire (and nerve-tire). The pain is increased on exertion, after mental, physical, or emotional effort. Neurasthenia, anemia, or local exhaustive disease (uterine, gastro-intestinal, etc.) is present. The muscles are usually flabby, and the vertebral column is not supported. The patient lounges or supports the back. Spinal curvatures are observed.

(b) Pain due to affections of the nerves. Nerve pain is recognized by the tender points and by vasomotor phenomena.

(c) Pain due to disease of the spine, the membranes, or the cord. (See above.)

Pain in the Extremities.—Pain common to all extremities may be due to (1) neuritis or neuralgia; (2) myalgia; (3) bone lesions; (4) joint lesions; (5) diseases of the bloodvessels: (*a*) intermittent claudication, (*b*) varicose veins, (*c*) prehemiplegic; (6) referred pain.

1. The pain of neuritis occurs in paroxysms, is aggravated by motion and associated with tenderness along the course of the nerve. It is usually unilateral. Neuralgias of an extremity are characterized by a dull ache, often with remission and exacerbation, and by the presence of tender points. Neuritis or neuralgia of the sciatic nerve or sciatica may be due to pressure on the sciatic nerve by a pelvic growth or fibroneuroma as well as an inflammation of the nerve itself. Bilateral pains of nervous structures may have a central origin, as spinal sclerosis, spinal tumor, or tabes. Pressure on the cord by new growths may also cause bilateral pains. Pressure on nerve trunks, as from a pelvic abscess or enlarged axillary glands, may cause bilateral pain in the first instance, unilateral in the second.

2. Muscle pains are diffuse, dull aching in character, exaggerated by motion, and usually alleviated by warmth. They may be bilateral, as (1) in the myalgia arising from unaccustomed exercise, rickets, scurvy, or acute infectious diseases; (2) in rheumatism or (3) in trichinosis. Unilateral pains may be due to strain or injury of the muscle. Temporary cramps of the muscles may be present after severe exercise. The cramps of the occupational neuroses, *e. g.*, writer's cramp, usually affects the upper limbs, and are induced by special acts or motions.

3. The pain of bone lesions is described as dull and grinding, exaggerated by motions and usually relieved by absolute rest, unless there is retention of pus when the pain is agonizing. The pain of a periostitis or osteomyelitis is exceedingly severe and accompanied by pronounced tenderness. Syphilitic bone disease is usually most painful at night and without the tenderness of other bone diseases.

4. The pain of acute joint disease is intense, agonizing, and always severely exaggerated by movement of the affected part. Gout is extremely painful and occurs characteristically in the smaller joints of the foot. Acute articular rheumatism and septic arthritis also cause severe pain, affecting usually the wrist, elbow, knee, or ankle. The chronic arthritides are usually accompanied by dull, often neuralgic-like pains, which are exaggerated by exposure to dampness and cold, and associated with contractures and atrophy of the muscles. Tuberculous joint disease is frequently painless. Pain due to strain of the ligaments of a joint is usually not severe and absent when the joint is immobile. Metatarsalgia (Morton's disease) is due to jamming of a nerve between the heads of the fourth and fifth metatarsal bones.

5. Pain, the result of disease of the bloodvessels, when it occurs, is habitually found in the lower extremities. Inflammation of a vein secondary usually to parturition or to typhoid fever is extremely painful at first, later dull and aching and associated with tenderness along the course of the vein. Spasm of an artery seen in sclerotic individuals,

or during muscular effort, causes a characteristic cramp-like pain—intermittent claudication. Cramp-like pains of an extremity, associated with formication and other paresthesias, is usually a premonitory indication of cerebral apoplexy. The early stages of local gangrene are also associated with pain.

6. Referred pain is much more common in the lower extremities than the upper. In the arms, particularly the left, shoots the pain of angina pectoris. To the thigh may be referred the pain of disease of the pelvic organs and bones, renal colic, or tumors of the lower abdomen; to the inner aspect of the knee, the pains of hip-joint disease; to the sole of the foot, the pain of acute prostatitis.

Pain in the external genitalia and the anus usually points to local diseases, though pain in the testicle or end of the penis may be present if there is ureteral colic.

CHAPTER V

DYSPNEA AND COUGH

DYSPNEA

DYSPNEA means difficult breathing. The respirations may be deeper than normal, or they may be both deeper and more frequent. The patient is usually conscious of suffering or of some distress in breathing. An extreme degree of dyspnea is called orthopnea; infrequent breathing, oligopnea, and simple rapid breathing, hyperpnea, polypnea, or tachypnea.

Dyspnea is recognized by the increase in the rapidity of the chest movements, with increased action of both the essential and the auxiliary muscles. When sudden in onset or extremely severe, as in orthopnea, the expression is characteristic. The *alæ nasi* move; the eyes and countenance are indicative of distress; and the pupils are dilated. As the dyspnea continues cyanosis develops.

Etiological Varieties of Dyspnea.—I. Lessened Amount of Air Supply.—

A. Obstruction of the air passages. B. Diminution of air space from (1) solidification; (2) compression; (3) impaired expansion.

A. OBSTRUCTION OF THE AIR PASSAGES.—1. *Pharyngeal Obstruction.*—Dyspnea is seen in tumors and in severe inflammations of the tonsils, in the rare form of erysipelas of the pharynx, and in retropharyngeal abscess.

2. *Laryngeal Obstruction.*—Dyspnea is a frequent as well as the most serious symptom of laryngeal disease. It may be due (1) to obstruction by inflammatory or edematous swelling, especially by a diphtheritic membrane; (2) to spasm; (3) to tumors or foreign bodies in the larynx; (4) to the cicatrization of ulcers after syphilis or lupus; (5) to paralysis of the abductors or adductors of the larynx. It may be, therefore, organic or spasmodic, and is inspiratory in type except in the more rare cases of a movable tumor below the vocal cords.

Dyspnea may vary in degree from slight inconvenience in breathing, noticed by the patient, to the violent struggling for breath that is seen in cases of extreme stenosis of the larynx. If carefully observed in either case, the larynx is seen to rise and fall.

Certain characteristic sounds attend the act of inspiration, depending on the nature of obstruction. In obstruction from simple spasm, or from intense inflammation of the larynx, without secretion, the sound of the inspiration is harsh and stridulous. In obstruction from edema or from exudation, as in laryngeal diphtheria, the sound of the inspiration is loud and stridulous, but not shrill. The expiration is usually noiseless

and prolonged. In spasmodic croup the expiration resembles snoring. The interval between expiration and inspiration is lessened; the respirations are hurried.

The dyspnea may be sudden in onset or may develop gradually. Acute paroxysms of dyspnea sometimes occur in the course of affections in which chronic dyspnea is present; thus sudden edema may occur in cases of syphilitic or tuberculous ulceration.

Diagnosis.—Laryngeal dyspnea must be distinguished from other forms of dyspnea: (1) Dyspnea from diseases of the heart and lungs. (2) Dyspnea from pressure upon the trachea. The larynx is not markedly moved during the respiratory acts, and the patient bends the head forward instead of backward. (3) Dyspnea from pressure upon the larynx. Cellulitis of the neck, tumors of the lymph glands, goitre, and retropharyngeal abscess are provocative of this form of laryngeal dyspnea. Dyspnea in diphtheria, frequently thought to be due to internal occlusion, may be due to pressure of enlarged glands on the bronchus and larynx.

3. *Tracheal Obstruction.*—The causes of tracheal obstruction are (a) external pressure; (1) tumor of the thyroid gland; (2) enlarged thymus; (3) thoracic aneurism; (4) mediastinal tumor from other causes than aneurism, as cancerous or tuberculous disease of the glands, or mediastinal abscess; (5) cancer of the esophagus; and (6) in rare cases, a dilated auricle. (b) Diseases of the walls of the trachea. They cause obstruction by narrowing the caliber. Syphilis is the most frequent cause of such obstruction. (c) Foreign body. The foreign body may remain free for a time, moving up and down as the patient coughs, and may at times be distinctly palpable. Later the foreign body usually becomes fixed in the right bronchus or in one of its main divisions, the opening of the right bronchus being more direct than that of the left.

In this form of dyspnea there is no increased movement of the larynx and no change in the voice. The dyspnea is inspiratory in type, and is never so extreme as that in laryngeal stenosis. A stridor attends the dyspnea, and is heard with the stethoscope over the trachea and over every part of the chest. Sometimes over the trachea a point can be isolated at which the sound is heard loudest, which point may indicate the seat of the stenosis. When the lower part of the trachea is obstructed, the sound is often more marked over the larynx than over the sternum. In the course of constant dyspnea paroxysms may occur during which the distress is unusually severe. The patient may complain of pain or oppression behind the sternum, or possibly only of slight soreness. Cough, with expectoration of mucus, usually attends the dyspnea. Sometimes the mucus is blood-tinged. If the obstruction is due to a foreign body, the dyspnea is of the same type, but occurs suddenly.

4. *Bronchial Obstruction.*—The causes of bronchial obstruction are: (a) *External Pressure.*—(1) Enlarged bronchial glands; (2) pericardial effusions and dilatation of the left auricle; (3) thoracic

aneurism; (4) mediastinal new growths; (5) mediastinal abscess; (b) bronchostenosis from disease of the bronchial wall, notably syphilitic ulceration; (c) foreign bodies. This type of dyspnea is not accompanied by increase in laryngeal movement or with change in the voice. When a bronchus is obstructed suddenly, compensatory emphysema occurs rapidly in the opposite lung; when gradually, it develops slowly, the degree depending upon the amount of obstruction. The physical signs over the cut-off lung are absence of the vesicular murmur and vocal fremitus, and impaired movement of the affected side. The percussion sound at first is normal, although it is influenced less by forced inspiration and expiration; later, it progresses from impaired resonance to dulness. As the case advances, the affected side may fall in and measure less than the opposite side. A snoring or whistling sound may be heard over the root of the lung, between the scapula and the vertebræ. Bubbling rales may be present.

B. DIMINUTION OF AIR SPACE.—Diminution of the air space lessens the available area for the free interchange of oxygen and carbon dioxide so that the carbon dioxide under tension is increased in the blood and there is a consequent overstimulation of the respiratory centres. Causes of this are:

1. *Compression of the alveoli*, as from congestion, edema, inflammations or morbid growths in the lungs. The degree of dyspnea depends upon the extent and suddenness of onset of the solidification.

2. *Compression from Intrathoracic Causes.*—Any variety of pleural effusion causes dyspnea from compression. It is more marked in bilateral than in unilateral effusions. Its severity depends somewhat upon the rapidity with which the effusion takes place. In cases of sudden effusion of air, as in pneumothorax, the dyspnea is very alarming at first, but is gradually relieved as accommodation takes place.

3. *Compression from Extrathoracic Causes.*—Pressure on the diaphragm as a result of increase of intra-abdominal pressure by tumors, ascites, etc., at times causes distressing dyspnea.

4. *Impaired Action of the Respiratory Muscles.*—(a) Weakness or paralysis of the muscles. Phrenic dyspnea is a peculiar form due to paresis of the phrenic nerve and consequent interference with the action of the diaphragm. It may not be observed so long as the patient is at rest, though the voice is weak and there is difficulty in coughing and sneezing, because a full inspiration cannot be taken. Instead of the natural expansion of the ribs and chest, the thoracic movements are reversed, and the shadow of the diaphragm cannot be seen. Dyspnea due to paralysis of the other respiratory muscles can be recognized by the atrophy of the groups of muscles concerned. (b) Inhibition of muscular action by pain. The seat of the pain may be in the pleura, in the muscles, or in the intercostal nerves. Frequently, as in peritonitis, hepatitis, etc., it is below the diaphragm, interfering with the action of that muscle. The dyspnea that occurs from pain resulting from pleuritis, or from inflammation of the chest wall, is

recognized by the posture taken by the patient in order to relieve the affected side, by local tenderness, and by the physical signs of pleurisy or of pleurodynia.

II. Lessened Amount of Arterial Blood in the Lungs.—Interference with the pulmonary circulation by anything that causes a decrease in the amount of blood carried to the alveoli, results in insufficient oxygenation of the blood and a consequent increase of tension in the blood of CO_2 , which acts as a direct stimulant of the respiratory centre. Such decrease may be caused by: (1) Tumors pressing on the vessels (rare). (2) Stasis in the vessels, as in unstable or non-compensated heart disease. Cardiac dyspnea is clinically divided into: (a) Dyspnea increased or caused by exertion. (b) Paroxysmal dyspnea. (c) Orthopnea. (d) Rhythmical dyspnea or Cheyne-Stokes respiration.

The dyspnea of effort comes on after the slightest exertion or excitement. In paroxysmal dyspnea the attack comes on without apparent cause. It must be distinguished from the paroxysmal dyspnea of uremia, asthma, and emphysema. Pulmonary disease can usually be recognized by the physical sign. The paroxysmal dyspnea of heart disease is attended by more violent respiratory efforts than the physical state of the lungs admits, and the difficulty attends both inspiration and expiration. Wheezing is not so marked as in certain forms of asthma. In paroxysmal dyspnea the breathing usually becomes quiet when the patient is placed in a comfortable position, provided there is no pulmonary or pleural complication. Posture does not modify the severe dyspnea of asthma or emphysema. Orthopnea and Cheyne-Stokes respiration are described later. (3) Thrombosis of pulmonary vessel. (4) Embolism, *e. g.*, fat. (5) Impaired quality or deficient quantity of blood, as in forms of anemia.

III. Interference with the Nervous Mechanism of Respiration.—The dyspnea may be of central or of peripheral origin, and may be due to various causes.

1. *Tumors, Hemorrhage, or Degeneration about the Respiratory Centre in the Medulla.*

2. *Irritation of the Centre by Toxic Agents.*—This may occur in uremia, in diabetes, and in infectious febrile conditions.

The nature of each variety is recognized more particularly by the associate symptoms. The dyspnea due to the poison of uremia usually occurs in paroxysms, but may be constant. The dyspnea of diabetic coma, known as "air hunger," is characterized by slow and deep inspirations, with short sighing expirations (Kussmaul's dyspnea). The dyspnea of infectious febrile conditions is due to the overfilling of the blood with carbon dioxide as a result of the increased metabolism during fever, to the action of the bacterial toxins and the increased temperature of fever, stimulating the respiratory centres.

3. *Reflex Stimulation of the Constrictor Fibers of the Pneumogastric Nerve.*—The constrictor fibers of the vagus that supply the bronchial muscles may be reflexly stimulated by some irritation to other fibers

of the nerve, causing in part the paroxysmal dyspnea of asthma. Such irritants may be (a) disease in the upper air passages, as polypus, hypertrophy of the turbinates, adenoid growths, and nasal congestion; (b) temporary irritants applied to the nares, such as various animal and vegetable odors, microorganisms or pollen in the inspired air; (c) disease in the fauces and larynx; (d) functional disturbances of the stomach and intestines.

Clinical Varieties of Dyspnea.—1. **Dyspnea Influenced by Exertion.**—

(a) *Shortness of Breath on Exertion Only.*—This is the form met with in anemia and in moderate cardiac insufficiency. It may not be observed by the patient unless he walks hurriedly or ascends a flight of stairs.

(b) *Shortness of Breath Independent of Exertion.*—This is of more serious import and may be due to a number of causes, but is usually seen in severe cardiac and pulmonary disease.

2. **The Respiratory Rate.**—Dyspnea varies clinically according to the frequency of the respiration.

(a) *Respiration Slow.*—In dyspnea the inspirations may be deep, and the frequency of respiration less than normal. The cases of such respiratory rhythm are usually either central (brain tumor, meningitis), toxic (autogenous intoxication as in uremia or diabetes or exogenous intoxication as in opium or chloral poisoning) or in the death agony.

(b) *Respiration Increased.*—The respiratory rate is increased in nearly all forms of dyspnea.

3. **The Respiratory Rhythm.**—Arrhythmic breathing. Two main types are seen: (1) Cheyne-Stokes breathing. (2) Biot's breathing. Irregular respirations conforming to no type occur in conditions of painful breathing.

4. **Constant and Paroxysmal Dyspnea.**—*Constant Dyspnea.*—This implies a persistence of the cause. It is frequently subject to paroxysmal aggravations. *Paroxysmal Dyspnea.* Paroxysmal breathing is seen in its most typical form in asthma and syphilitic aortitis.

5. **Inspiratory and Expiratory Dyspnea.**—Dyspnea is divided into inspiratory and expiratory dyspnea, according as there is interference with the inflow or outflow of air. Obstruction to the larynx, trachea, and bronchi are the causes of the former. Emphysema, asthma, and chronic bronchitis are causes of the latter type of dyspnea.

6. **Subjective Dyspnea.**—The respirations may be hurried and create distress in simple nervousness and hysteria without any obvious cause for the condition.

COUGH

Coughing is a reflex act. Cough may be due to (1) irritation of the respiratory tract and to (2) reflex irritation.

1. **Irritation of the Respiratory Tract.**—This usually begins in the respiratory mucous membrane, the purpose of the cough being to expel accumulations of mucus or pus, or some foreign substance. It

occurs in all forms of bronchitis and in lung affections associated with bronchitis. The cough of phthisis when not laryngeal is due to a localized bronchial catarrh. Cough is notably present in pharyngeal and laryngeal diseases. (See Diseases of the Nose and Larynx.) The presence of an irritant does not always excite cough, when the sensibilities are obtunded, as in typhoid fever, in disease of the brain, or in the last stages of any disease. In phthisis sudden cessation of the cough and expectoration from weakness is a bad prognostic sign and denote approaching death. It is also a bad sign in pneumonia.

2. Reflex Irritation.—There are several varieties of cough due to this cause.

(a) **Nasal Cough.**—(See Nose.)

(b) **Pharyngeal Cough.**—This may be due to hypertrophy of adenoid tissue, inflammation, paralysis, or new growths of the pharynx, to an elongated uvula, or to the presence of secretion from the nose.

(c) **Pleural Cough.**—This is not of infrequent occurrence and is due to acute or chronic inflammation of the pleura. The so-called "live cough" accompanying hepatic enlargements at times, is probably due to irritation of the diaphragmatic pleura.

(d) **Ear Cough.**—The most characteristic cough of this form is that due to the presence of a foreign body in the meatus of the ear; or to disease of the ear. According to Fox, the efferent nerve that transmits the irritation is the auriculotemporal branch of the fifth nerve.

(e) **Dentition.**—In infants, dentition and in adults the irritations of the stump of a tooth may cause cough.

(f) **Miscellaneous Causes.**—Cough may accompany cardiac disease. It may exist with enlargement of the bronchial glands, with mediastinal tumors or abscess, with thoracic aneurysm, and with carriers of the dorsal vertebræ, all of which, however, probably cause some secondary intrapulmonary condition. It may accompany disease of the stomach and is due to an associated secondary pharyngitis.

Clinical Characteristics of Cough.—The cough may be dry (unproductive), moist (productive), constant, or paroxysmal.

Dry Cough occurs when the source of irritation is not removable. It is seen in the first stage of pneumonia and bronchitis, and in the earlier stages of phthisis. In pleurisy the cough is short, hacking, and suppressed. It occurs also in laryngitis, tracheitis, bronchial asthma, influenza, and pertussis. It is the type met with when the cough is due to irritation outside of the respiratory tract entering the tract, and is excited by the inhalation of dust, irritating fumes, and tobacco smoke.

Moist Cough is attended with the production of sputum of a mucopurulent, purulent, or bloody character, which is removed with comparative ease.

Constant Cough usually implies a persistence of the cause, which is invariably pulmonary or bronchial, as pleurisy, phthisis, bronchitis, and solidifications. The cough is almost constant when the irritation

is permanent, and when a large amount of secretion is rapidly being poured out. The latter is seen in bronchorrhea, in bronchial dilatation, and in the later stages of tuberculosis.

Paroxysmal Cough occurs with suddenness and intensity and persists until removal of the irritant. It is the cough that occurs typically after the insufflation of water or some foreign body. In the second stage of bronchitis, paroxysms of cough may occur every few hours, or only once in twenty-four hours, usually in the morning on arising. Paroxysmal coughs occur in cases of bronchorrhea, bronchial dilatation, pulmonary abscess, gangrene or cavities, either of the lung or of the pleura opening into the lung, being excited whenever the cavity fills with secretion. The paroxysm may occur daily or several times a day, and an enormous amount of sputum is thrown off, until the cavity has been emptied. In these affections the cough is further characterized by the fact that it is aggravated by change of position. The association of retching and vomiting with the paroxysm is of some diagnostic significance, and is seen not only in whooping cough, but also in phthisis. In pertussis the character of the paroxysm is of special diagnostic significance.

Laryngeal Cough.—This is a constant accompaniment of diseases of the larynx. Several forms are noted:

1. The *dry cough* as seen in acute laryngitis. It is almost constant, and is aggravated when the patient speaks, takes fluid, or inspires deeply. In children it is abrupt, brassy or metallic, stridulous or whistling, so-called "croup-cough" as seen in cases of "false croup" and laryngitis with edema.

2. A dry, hoarse cough occurs in the course of chronic laryngitis.

3. The cough is of such a character as to give one the idea that it is suppressed in membranous and edematous laryngitis.

4. A cough frequently occurs without any local anatomical changes in the larynx, which seems to be purely of nervous origin. Two forms are seen:

- (a) *Paroxysmal*.—Severe coughing occurs suddenly, and cannot be controlled by the patient.

- (b) *Continued and Rhythmical*.—The cough is not so severe as in the paroxysmal form, but also occurs at more or less regular intervals, and varies somewhat in intensity from time to time. It does not occur during the act of eating or speaking, and ceases entirely during sleep. It is usually worse when the patient is under observation. Examination with the laryngoscope reveals absence of disease. Nervous cough is seen after diphtheria, when sexual disturbances are present, at puberty, and in some cases of anemia, chlorosis, neurasthenia, and hysteria. The pitch is usually high.

5. A *brassy, metallic cough* is frequently caused by irritation of the recurrent laryngeal nerve by a thoracic aneurism, a mediastinal tumor, or by enlarged bronchial glands.

Diagnostic Value of Cough. The diagnostic value of cough is estimated by the character, whether dry or loose, whether constant or paroxysmal; by the sound, which, however, is usually modified by the condition of the larynx (see Section on Laryngeal Diseases), and by means of which laryngeal is distinguished from bronchial disease; by its frequency, by its duration, and by its development at particular times or under particular circumstances, as on rising in the morning, or on changing to a cold atmosphere, or in speaking, or upon movement. The diagnostic value of cough further depends on a knowledge of the character of the expectoration (see Sputum).

CHAPTER VI

DYSPHAGIA—VOMITING—CHANGES IN APPETITE— HICCOUGH

DYSPHAGIA

DIFFICULT swallowing is due to a number of causes, the chief of which are diseases of the mouth and fauces, disease of the larynx, and disease of the esophagus.

Glossitis, cancer of the tongue, the various forms of stomatitis, inflammations of the tonsils, and all forms of pharyngitis cause painful swallowing. The mouth eruptions of the eruptive infectious diseases may cause it. Rheumatism of the pharynx and retropharyngeal abscess are causes that occasion the greatest difficulty in diagnosis.

Difficulty in the act of swallowing in laryngeal diseases is most marked when tissue destruction in the larynx has taken place, or when there is acute inflammation about the muscles or their attachments; hence, when tuberculosis or malignant ulcers are present, or perichondritis arises, the difficulty is so great as to prevent the taking of food. There is great dysphagia when the epiglottis is the seat of acute inflammation. Laryngeal dysphagia is recognized by pain and by the falling of particles of food into the larynx, exciting cough.

Dysphagia is a symptom of all diseases of the esophagus. It may vary from simple painful swallowing to complete obstruction of the tube. It may be due to paralysis, to spasm (esophagismus), or to obstruction.

Dysphagia from obstruction of the esophagus or stricture is due (1) to disease outside of the canal (external pressure); (2) to disease of the canal itself, and (3) to the presence of a foreign body in the canal.

1. **External Pressure.**—The esophagus at different parts of its course is in intimate relationship with the trachea, the thyroid gland, the carotid artery, the left bronchus, the bronchial glands, the arch and the descending portion of the aorta, so that disease of these structures may result in dysphagia.

Disease of the trachea, thyroid gland, or carotid artery is readily recognized. Within the thorax disease of the mediastinal glands, aneurism of the arch or descending portion of the aorta, an enlarged left auricle, a pericardial effusion, or disease of the left bronchus which might cause constriction of the esophagus is recognized by the symptoms and physical findings of these conditions.

2. **Organic Disease.**—Difficulty of deglutition due to disease of the esophagus itself occurs in acute inflammation, in chronic inflammation and in stricture which is the result of syphilis, cancer, or inflammation as from ingestion of caustic alkali or acid.

3. **Foreign Body.**—Stricture from the presence of foreign bodies is usually recognized with ease. The difficulty of deglutition is due both to the foreign body and to the spasm it excites. In consequence of the latter, regurgitation of food takes place. In the first place there is a history of the swallowing of a foreign body. Sudden pain succeeds the act, while there are dyspnea, great anxiety, and distress, particularly if the body is a large hard mass. If the obstruction cannot be removed, ulceration and abscess result, the further course of which depends upon the seat of the obstructing material. Pain, hemorrhage, subcutaneous emphysema, and the emission of air are the symptoms that may follow. The exact location of the foreign body may be ascertained by the use of the Röntgen rays, the esophagoscope, or esophageal bougies.

VOMITING

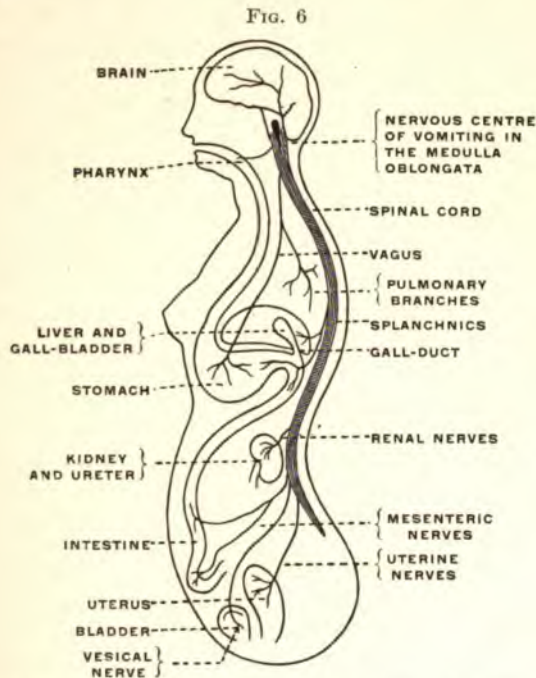
During the act of vomiting the stomach is compressed by the abdominal muscles and diaphragm, while the so-called cardiac sphincter of the esophagus is at the same time relaxed. Both factors must operate at the same time to produce vomiting, which explains why it is that some persons suffer extreme nausea and even have violent retching but are unable to vomit. The nervous centre of vomiting is seated in the medulla oblongata in close proximity to, and immediately connected with, the respiratory centre. It is to this centre that impressions are sent from the brain itself or from various portions of the body through their nerve supply, and from this centre motor impulses are transmitted to the muscles concerned in the act of vomiting and to the stomach and esophagus.

By a very good diagram (Fig. 6) Brunton indicates the afferent nerves which transmit impulses to the vomiting centre and excite it to action. Fibers also pass downward from the brain, transmitting impressions to the vomiting centre from the organs of special sense from the brain substance or its membranes when they are diseased or from central ganglia excited by emotion or imagination.

From this it is seen that vomiting is a reflex act, but reference has not been made to the vomiting that occurs in the initial stage of many fevers and in septicemia, uremia, and allied affections. These are doubtless due to the direct action of toxins in the blood on the vomiting centre.

In seeking for an explanation of vomiting it is of importance to find out the previous state of health of the patient—whether the vomiting occurred after the patient has been ill for a longer or shorter time or suddenly when he was in apparent health; or whether it formed one of the initial symptoms of an acute disease.

Inquiry should be made as to the supposed *cause* of the vomiting; whether it was excited by the taking of food, or medicine, or by some disgusting sight or odor. The *time* when the vomiting occurred should be ascertained, as well as its frequency, and whether it was preceded by nausea, pain (noting its locality), injury, cough, jaundice, or constipation. The *position* of the patient at the time of vomiting sometimes furnishes a valuable clue to its cause. The *effect* of vomiting is sometimes of aid in diagnosis. In ulcer and in migraine, for example, it affords marked relief. Finally, the *appearance* and *quantity* of the matter vomited are very important subjects for investigation. (See Objective Signs.)



The nervous mechanism of vomiting. (After Brunton.)

Character.—Vomiting may occur occasionally, persistently, or periodically. It may be projectile and painless, or difficult and painful. Projectile vomiting is characteristic of cerebral disease or points to a reflex origin. The latter type indicates local gastric disease. Sudden vomiting without antecedent illness usually indicates some local affection of the stomach, or is due to some nervous impression, or marks the onset of some acute general disease, as in pneumonia, the eruptive fevers, and yellow fever. Excessive vomiting generally indicates that the illness will be severe.

Vomiting in Gastric Diseases.—The local affections of the stomach attended by vomiting are acute and chronic gastritis—especially the catarrhal form, ulcer, cancer, dilatation, and pyloric obstruction.

In *acute gastritis* there will be a history of an acute illness marked by severe local and general symptoms. The vomiting is preceded by nausea and epigastric pain and tenderness and is often followed by profound prostration. In *chronic gastritis*, vomiting often occurs in from half an hour to an hour and a half after eating. It does not produce the prostration incident to vomiting in acute gastritis, and is followed by some relief of the gastric uneasiness and pain. In *ulcer* of the stomach vomiting is usually absent. Occurring usually soon after the taking of food, it relieves the gastric pain, but there is nothing characteristic in the vomit unless it contains food. In *cancer* of the stomach vomiting is an almost constant symptom, in some instances one of the earliest symptoms. There is no uniformity in the frequency of its occurrence nor in the character of the vomit. In *dilatation* of the stomach with or without pyloric obstruction, which, however, is always associated sooner or later with dilatation, vomiting may be deferred for several days and then be correspondingly more copious.

Vomiting in Other Conditions.—Nausea and vomiting are excited in some persons by the sight of blood or by a horrible or loathsome spectacle; others are more susceptible to foul odors and disgusting tastes.

Again, irritation at some extragastric point is transmitted by the proper afferent nerve to the vomiting centre and then reflected to the stomach. Vomiting of this character occurs in pregnancy, and in diseases of the vermiform appendix, ovaries, uterus, bladder, prostate gland, lungs, nose, eyes, kidneys, intestine, peritoneum, liver, pancreas, gall-bladder, and bile ducts, particularly when associated with severe pain.

The particular organ that is the source of the irritation must be determined by a careful physical examination guided by the indications furnished by age, sex, time of occurrence, habits, and the symptoms that accompany the vomiting.

Vomiting in Toxemias.—Vomiting is also a prominent symptom of toxemias, being probably produced by direct irritation of the vomiting centre by the toxins. Among such disorders are the specific fevers, notably scarlet fever and yellow fever; sewer-gas poisoning; pregnancy; and diseases of the liver and kidney which produce cholemia and uremia, particularly cirrhosis of the liver and interstitial nephritis.

Cyclic Vomiting.—Periodic vomiting in children, due possibly to an acid intoxication and recurring at regular intervals, has been described by Leyden. It is sudden in onset, severe, and accompanied by retraction of the abdomen and great prostration.

Cerebral Vomiting.—Vomiting due to cerebral disease is a well-recognized symptom; in early life it is a characteristic feature of meningitis and tumor of the brain, and is likewise of moment in later

life. A sudden, violent expulsion of the stomach contents, ceaseless, unrelieved by remedial measures, has been seen by the writer to precede other signs of apoplexy by from thirty minutes to twenty-four hours. In all cases of apoplectic character the pulse is slow and full, while in nausea and vomiting from other causes, in the aged particularly, it is weak and feeble. Moreover, some alteration of breathing is noticed. It is either irregular, or slow, or unduly hurried, and proves the intimate relation of the vomiting to the respiratory centres. Further, strength is seen, not weakness; in the apoplectic the face is congested, not pallid as in simple sick stomach. The other peculiarities of cerebral vomiting have been indicated.

Crises.—Sudden attacks of vomiting with hyperacidity, with or without pain, often occur in locomotor ataxia and in other affections, as hysteria.

Diagnosis.—Vomiting is readily recognized. However, it is often productive of serious lesions. It may cause apoplexy or cerebral congestion; or acute overdistention of a dilated heart, as in aortic regurgitation. If it continues for any length of time, and much fluid is ejected, vomiting is attended by anuria and rapidly followed by collapse. It also induces thirst.

CHANGES IN THE APPETITE—THIRST—FLATULENCE— HICCOUGH

The following subjective symptoms may be complained of, and while they are common in gastric disorders, may be present in other general and local diseases: disorders of appetite, bad taste in the mouth, thirst, eructations, pyrosis, distress or weight after meals, burning after meals, flatulence, nausea, vomiting, constipation, diarrhea, pain, vertigo, and cardiac palpitation. Nearly all these subjective symptoms represent gastric disorders, and will be more fully described in the section devoted to that subject.

Bad Taste.—This is usually due to acute catarrh, but may be present in chronic catarrh. It may be due to local causes, as decayed teeth, disease of the mouth, tonsils, or nose. It may be due to medicines—*e. g.*, potassium iodide—or to metallic poisoning.

Thirst.—Thirst is not a symptom of gastric disorder alone; it is a symptom of diabetes and all conditions in which the body has lost fluids, by sweating, vomiting, or purging; by evaporation and combustion (fever) or by hemorrhage. It is common in acute and chronic gastritis, particularly in the alcoholic forms.

Distress, Weight, and Burning.—These complaints are very common and may come on immediately after meals; they may be due to dyspepsia, hyperacidity, dilatation, fermentation, or flatulence, and exist in varying degree, either singly or combined. (See Gastric Hyperesthesia.)

Nausea.—This symptom is usually associated with vomiting. In some persons it is impossible to excite vomiting, although they may suffer intolerably from nausea. Nausea is akin to vomiting in its mechanism and clinical associations (*q. v.*).

Alterations of Appetite.—*Anorexia*, or loss of appetite, may be due to a number of causes. It is present in all forms of organic disease of the stomach except occasionally in ulcer. The appetite may or may not be impaired in gastric neuroses. Anorexia is of frequent occurrence in disorders remote from the stomach that modify the condition of the organ reflexly. In the section on Vomiting will be found statements showing the influence of central disease. Loss of appetite is a constant accompaniment of the moderate gastritis which attends all fevers. It is seen in dyspeptics who have carried out a restricted dietary—*anorexia* from disuse. In hysteria and neurasthenia it is fairly common, and when extreme is known as *anorexia nervosa*. In all forms of anemia, in toxic conditions, in all chronic wasting diseases, in chronic infections, in septic conditions, and in many functional and organic diseases of the nervous system the appetite is lost.

Boulima, or excessive appetite, is a common symptom in the earlier periods of diabetes, and is said to be present in disease of the mesenteric glands. It occurs also in gastric neuroses. Perversion of the appetite, in which all sorts of substances are greedily swallowed, occurs in hysteria, dementia, and pregnancy.

Flatulence, Regurgitation, Belching, and Pyrosis (Water Brash) are common symptoms of primary or secondary gastric disorders, though they are occasionally seen in conditions such as hysteria.

Hiccough.—Hiccough, or singultus, is a spasm of the diaphragm. The contractions take place at more or less regular intervals, and are attended by a peculiar clicking sound, due to the sudden passage of air through the glottis. Hiccough may be a serious symptom; it may last for a few minutes or continue for several days. In the latter case it causes extreme exhaustion. In chronic disease its occurrence is of bad prognostic omen. It may be due to exhaustion alone, as in the typhoid state or in shock; to central nervous disease, as brain tumor or meningitis; to hysteria and mental emotion; it may be the result of toxemia, as in uremia, diabetes, or gout; or it may be of purely local origin, as in gastric carcinoma, intestinal obstruction, peritonitis, and hepatic and pancreatic disease.

CHAPTER VII

DIARRHEA AND CONSTIPATION

DIARRHEA

DIARRHEA is an increase in the frequency of the bowel movements, the stools usually being small and watery. It may vary from a transient increase in the number of daily movements to the continuous purging of cholera. Diarrhea is due to increased peristaltic activity of the intestine, to an increase of intestinal secretions, or more frequently a combination of the two. The following classification of diarrhea may be recognized:

Nervous Diarrhea.—Increased peristalsis may be due to some impression upon the nervous mechanism of the intestine. This may explain the diarrhea of emotion, or that which occurs from other psychical influences. A similar condition is seen in the attacks of diarrhea which occur in tabes. The diarrhea that accompanies mucous colitis, a secretory neurosis, is due in part to the increase of the intestinal secretion.

Catarrhal Diarrhea.—In the larger number of cases the diarrhea is due to catarrhal inflammations of the intestinal tract. The causes of the catarrhal inflammation are many, and have been divided into primary and secondary causes.

Primary catarrh is due to the direct influence of causal factors upon the mucous membrane. Its causes are practically those of acute enteritis, and include the direct irritation of undigested food, from the action of irritants, as of bacteria or their products, and the action of certain poisons, such as mercury, arsenic, and other corrosive agents. The diarrhea which occurs from the irritant action of food products and in cholera infantum is due to a toxic ptomain or to actual infection. Catarrhal inflammations due to microorganisms is the most frequent form in children. *Secondary* catarrhs follow other lesions, as ulcers. The catarrh, and hence the diarrhea, that attends the ulceration of typhoid fever and of dysentery; that which occurs in the course of Bright's disease; and the diarrhea that attends carcinoma or other organic disease of the bowel, are of this nature. In addition, a catarrh of the bowels may arise from venous stasis in the mucous membrane from chronic congestion, as in organic heart disease with congestion of the liver, from amyloid disease or from the secondary irritation that occurs in extensive burns.

Spurious Diarrhea.—This is the diarrhea of constipation. Fecal impaction may be associated with diarrhea. The mass of feces may be

channelled or a colitis may occur secondarily. The evacuations are scybalous, coated with mucus, and often bloody. Tenesmus is present.

Vicarious Diarrhea.—Diarrhea sometimes fulfils a vicarious office. This is the case with the diarrhea which comes on in cases of chronic and acute Bright's disease before the supervention of uremia. When diarrhea occurs in a person with pallor, dimness of vision, and edema, the urine should always be examined.

Symptoms.—The symptoms that attend diarrhea depend upon the cause and the frequency of the evacuations. The most frequent symptoms are pain and flatulent distention with borborygmi and tenesmus.

Increased Movements of the Bowel.—The frequency of the movements varies with the cause and its severity.

Pain.—The pain depends largely upon the cause. When the irritant is a product of indigestion or a bulky mass, pain is more or less severe and situated in the centre of the abdomen, or diffuse. The pain occurs before defecation; it is sharp, lancinating, and is usually relieved by the act. When the inflammation is in the large intestine, the pain may be complained of in the course of the large bowel or be more intense over the cecum and the sigmoid flexure. The rectum may be the seat of pain or of painful sensations, as if there were a hot ball in the lower pelvis.

Flatulent Distention.—The flatulent distention is usually not very great. With the distention occur borborygmi, which usually subside after the evacuation.

Tenesmus, painful, ineffectual straining at stool, occurs in all forms of diarrhea when the evacuations are frequent. In severe cases it may be almost continual, and may lead to prolapse of the bowel. It is of common occurrence in prostatic enlargements, pelvic growths, rectal diseases, vesical calculus, impacted feces, worms or foreign bodies in the rectum—conditions which must be excluded.

Prostration.—More or less prostration attends all cases. It is, however, more marked as a result of frequent watery evacuations.

Character of the Stools.—1. The stools are *fecal*, with a small amount of water. They are light in color, softer than natural, but yet retain their form—the kind of movements seen in simple catarrh.

2. The fecal matter is mixed with *undigested food*. The feces are in scybalous masses, and the watery element is increased. They are the stools of the so-called dyspeptic diarrhea.

3. Along with the feces more or less *mucus* is seen. Inflammations of the large intestine are attended with mucus discharge, which may be mixed with and stained by feces so that it can be recognized only by close inspection. In milder degrees of catarrh mucus is seen on the surface of the fecal masses.

4. Formed feces disappear almost entirely, and instead the evacuations are *watery*. The watery stools may be discolored, as in the pea-soup evacuations of typhoid fever; or they may be almost clear water, as in the rice-water discharges of cholera.

5. The evacuations may contain *blood*. Bloody discharge usually accompanies the discharge of mucus; when the catarrh is in the lower bowel, blood may occur independently of mucus and small amounts of free blood are seen. When both are present, the mucus is tinged with reddish specks. The blood may be bright in color, and then usually comes from the rectum (hemorrhoids). If mixed with the stool the blood may be black, as in all forms of melena, or it may be dark red in color. Black blood usually comes from the stomach or the first part of the duodenum, and may be the result of ulceration, or even the swallowing of blood.

Microscopic Examination.—(See Feces.)—In simple catarrhal inflammation but little is found on microscopic examination except an excess of epithelium from the mucous lining. In more intense inflammations in addition to epithelium pus and blood and mucus are found.

Chronic Diarrhea.—Chronic diarrhea may be due to chronic inflammation of the bowels, as in chronic intestinal catarrh. It may be secondary to the ulceration of dysentery, tuberculosis, syphilis, or cancer. It may be secondary to chronic gastritis with anacidity, or attend the neurôses of gastro-enteroptosis. It is the common diarrhea of amyloid disease. In chronic diarrhea the number of stools varies, but seldom amounts to more than ten to fifteen a day. In chronic intestinal catarrh three or four movements occur in the twenty-four hours, the first evacuation taking place immediately on rising, and the remainder during the morning hours. Women suffer more than men in this respect, the movements being readily excited by exhaustion or nervous influence, as grief, emotion, or excitement of any kind. The stools are watery and contain fecal matter, which is usually coated with mucus. The color of the feces is not changed. The patients usually suffer from intestinal dyspepsia, or they are subject to some gastric neurosis. They are not under-weight, and except for the inconvenience of the morning hours, quite equal to the ordinary demands of life. They are more nervous than most people and are liable to attacks of hemicrania.

CONSTIPATION

Constipation may be due to a number of causes, general and local.

General Causes.—These include alteration or diminution in the secretions of the intestinal tract, as is seen in all fevers or in the summer when there is free perspiration and is present in affections attended by exhaustive diuresis as in diabetes. Diminution in the sensibility of the nerves may exist. This is the one chief cause of habitual constipation, which is so prevalent. Through carelessness the patient loses the habit of having a regular movement of the bowel each day and in consequence the usual stimulus is removed. Again, the excessive taking of purgative drugs causes secondary constipation, the result of overstimulation and subsequent failure of response to normal

stimulation. A sedentary life as well as improper diet of too little food and food without residue may produce constipation; a family tendency to constipation may be present.

In anemia and chlorosis, in neurasthenia and hysteria, cachexia and debility, in the convalescence from exhausting disease and prolonged confinement to bed, in chronic diseases of the stomach, heart, liver and kidneys, in meningitis and myelitis, the constipation may depend upon atony of the musculature of the intestines, alteration of the intestinal secretions, or impairment of the nervous mechanism.

Local Causes.—These include weakness of the abdominal wall or diaphragm from overdistention by tumors, repeated pregnancies, ascites, and obesity; atony of the intestines, which may be a result of any of the general causes of constipation; paresis of the muscular wall of the intestines, as in peritonitis; pathological states of the intestinal mucosa from chronic inflammations or congestions; anatomical defects in the intestine, as kinks at the junction of the duodenum and ileum, at the lower end of the ileum and at the hepatic and splenic flexures of the colon, a redundant sigmoid, ptosis of the colon, congenital dilatation of the colon, etc.; deficiency in the intestinal secretions or in the secretion of bile; lack of fecal matter the result of esophageal obstruction (stricture or cancer) or pyloric obstruction (stricture, spasm from hyperacid irritative gastric secretions, malignancy, etc.); intestinal obstruction (*q. v.*); interference with propagation of peristalsis by pyloric disease or by bands of adhesions which are, however, not sufficient to cause complete obstruction; spasm of the large intestine seen in nervous and hysterical women, or of the sphincter ani the result of painful fissures or hemorrhoids, and the action of such drugs or poisons as opium and lead or astringents generally.

Symptoms.—Constipation is characterized by diminution in the amount or the frequency of the bowel movements. The frequency of the movements varies in health. Some persons are comfortable with an evacuation once a week or at most every third or fourth day. Others may have daily movements, but the stools are small and scanty, with an occasional passage of large amounts of fecal matter. While in many instances symptoms are of no consequence, in others the patients are nervous and may be in a more or less impaired state of health from the secondary effects. The patients are of spare habit, and usually of dark or muddy complexion. They may be depressed and more or less hypochondriacal, and there is inaptitude for mental exertion. Dull headache and vertigo are common. The tongue is constantly furred, the appetite variable; a feeling of weight and fulness after eating and some degree of flatulence and borborygmi are usually present; rectal discomfort may be noticed.

The Secondary Effects of Constipation.—The effects of constipation upon the intestines are various and sometimes disastrous. They are dilatation and ulceration. The dilatation may be so great as to distend the entire abdomen. Ulceration may be localized to the rectum or

cecum, or extend throughout the entire large intestine, and may at times lead to perforation of the intestines. Hemorrhoids are very frequently secondary to chronic constipation. Chronic gastric catarrh is another result which frequently develops.

Incontinence of Feces.—This may arise because the centres controlling the act of defecation are impaired by disease of the lumbar cord. Afferent fibers from the rectum pass to centres in the lumbar cord, from which efferent fibers return to the sphincter ani. Through higher centres, as in the brain, the lumbar centre may be inhibited. When the rectum is full, impulses are sent to the lumbar centre and the brain, and unless they are resisted by the will, the act of defecation takes place reflexly. Thus, incontinence of feces may occur in the insane, in delirious persons, in coma, and in profound prostration. It may also be due to paralysis of the sphincter or to injury of the pelvic floor.

Painful defecation is usually attended by bloody stools when an anal fissure is present. Hemorrhoids and enlarged prostate and prolapse of the rectum give rise to pain. Disease of the pelvic viscera may also cause painful defecation.

CHAPTER VIII

THE VOICE AND SPEECH

APHONIA

THE most common symptom of affections of the larynx is alterations in phonation through disturbance of the function of the vocal cords. The voice may be hoarse in acute or chronic inflammations and congestions, in tumors, in specific ulcerations about the larynx, and in paralysis of the cords. From simple hoarseness it may vary in intensity to complete loss of voice (aphonia).

Chronic Hoarseness.—This may be due to chronic laryngitis. Slight hoarseness deepening to aphonia, attended by soreness, and later some dysphagia, is seen in tuberculous laryngitis. The duration may be significant. Hoarseness of long duration (years) is said to be prodromal of cancer (Ziemssen).

Functional Aphonia.—This may occur after excessive use of the voice and in hysteria. Hysterical aphonia occurs in women and young girls; the laryngoscope reveals nothing; the acts of coughing, laughing, and sneezing are normal, and a sound may be created in either act; it appears and disappears suddenly.

DISTURBANCES OF SPEECH

Disturbances of speech may be divided into two groups: aphasia, the disturbance of the central nervous mechanism controlling speech, writing, and mimicry; and anarthria, the disturbance of the peripheral motor mechanism of speech.

Aphasia.—By aphasia is meant the loss or impairment of the ability to understand spoken, written, or mimic language, and to express thoughts by the same means. It is divided ordinarily into two forms: motor aphasia, or the inability to innervate the motor apparatus for speech, while the sensory or perceptive functions are intact; and sensory aphasia or the inability to recall or understand words, although the ability to produce sound is preserved. A variety of other forms, however, have in the course of time come to be recognized. Oppenheim recognized the following five varieties:

1. **Motor Aphasia.**—This consists of the loss of power to speak, with persistence of the understanding of spoken, written, and mimic speech. The lesion is cortical or subcortical, and involves the foot of the third frontal convolution on the left side.

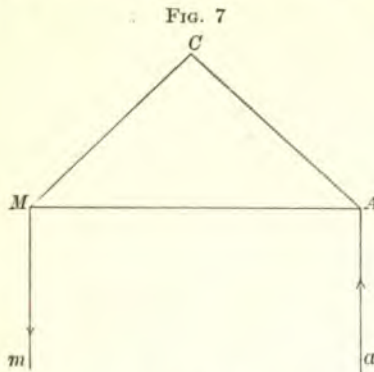
2. **Sensory Aphasia.**—The perception of sound as such is preserved, but there is inability to recognize the significance of words. The lesion is found usually in the auditory centre, that is, the first temporal convolution on the left side.

3. **Pure Alexia or Word Blindness.**—Although sight is preserved and objects may be recognized, the ability to understand written or printed language is lost. The lesion is found usually in the left occipital lobe on the external surface, but sometimes involves the gyrus angularis.

4. **Pure Agraphia.**—This is loss of the power of writing, all the other qualities remaining normal. Lesions have been found in the left upper parietal lobe.

5. **Optic Aphasia.**—Objects may be seen and recognized, but it is impossible for the patient to find the proper name for them. The lesion is found usually at the junction of the first temporosphenoidal and the occipital lobes.

In order to test the aphasia it is necessary in each case to determine whether the following normal functions are present or absent: (1) Voluntary speech; (2) the ability to repeat words; (3) reading aloud; (4) voluntary writing; (5) writing from dictation; (6) copying; (7) speech perception; (8) understanding of writing; (9) the existence of paraphasia.



In order to explain aphasia, it has been customary, since the time of Wernicke, to employ the diagram shown in Fig. 7. The triangle ACM represents the intracerebral paths and centres for the mechanics of speech and the lines Aa and Mm the peripheral apparatus. A represents the centre for auditory perception; M , the centre for the emission of motor impulses, and C the concept centre, in which the intellect analyzes the impressions received and from which the directing influence for the choice of language is transmitted to the motor centre. Aa represents the auditory nerve; Mm the motor nerves to the pharynx, tongue, and lips. Auditory impressions may, therefore, be transmitted along Aa to A , thence directly to M .

and thence to the larynx. This is the mechanism supposed to be involved in ordinary mechanical speech, that is to say, the mechanical repetition of spoken words. The auditory impressions may, however, pass from *A* to *C*, there to be analyzed or understood, and then transmitted to *M*, either in the same or in an altered form. This constitutes the intelligent repetition of spoken languages. If the alteration of form is considerable, or if, without immediate auditory impressions, impulses are transmitted from *C* to *M*, voluntary or intelligent speech is said to occur. The varieties of aphasia that can theoretically be deduced from this diagram correspond more or less closely to those that may be recognized in actual practice. In general, lesions in the centres themselves cause *cortical* aphasia; in fibers connecting the centres, *transcortical* aphasia; in those running back and forth from the lower ganglia and nuclei, *subcortical* aphasia. More specifically these varieties are as follows: Destruction of the motor centre *M* gives rise to the so-called cortical motor aphasia with the following symptoms: loss of (1) voluntary speech; (2) repetition; (3) reading aloud; (4) voluntary writing; (5) writing from dictation. Destruction of the auditory centre *A* gives rise to cortical sensory aphasia. There are lost (1) the understanding of speech; (2) the understanding of writing; (3) the ability to repeat speech; (4) the ability to write from dictation; (5) the ability to read aloud. A lesion at *C* would give rise to cortical apperceptive aphasia. The symptoms of this form would differ very slightly from those due to interruption of the tracts supplying it. The centre is probably complex and its parts are widely distributed. The speech-disturbances of general paresis are due possibly to partial destruction of the concept centre. Lesions of the various tracts of fibers connecting the different centres with each other or with the periphery also produce symptoms. Lesions between *A* and *M* produce the symptoms known as *paraphasia*. The only symptom of this condition is the misuse or false pronunciation of words. Thus objects may be misnamed, one word used in place of another, different syllables of the words misplaced (literal aphasia) or the words jumbled in a sentence (verbal aphasia). There is usually also *paragraphia*, that is, a similar disturbance of written language; *paralexia*, manifest when the patients attempt to read aloud; and sometimes the symptoms known as *agrammatism*, that is, the misuse of cases, moods, or tenses. Paraphasia, however, occurs also in certain general diseases of the brain, and is practically always present if the intrinsic tracts concerned in speech are disturbed. Interruption of the tract uniting *C* and *M* causes transcortical motor aphasia. There are lost (1) voluntary speech and (2) voluntary writing. The most characteristic symptom is the inability of the patient to remember words, although he is able to repeat them fluently. The interruption between *A* and *C* gives rise to transcortical sensory aphasia. There are lost (1) the understanding of speech; (2) the understanding of writing. Usually both voluntary speech and writing are affected by the paraphasia resulting from the

interruption of the intrinsic tracts. It differs from the preceding form particularly in the fact that the meaning of words spoken upon repetition or written from dictation is not grasped by the patient. In this form, communication with the patient, even by gestures, is often impossible. Finally, lesions may occur in the tracts uniting the centres concerned in speech with the periphery. Lesions in the tract *Mm* give rise to subcortical motor aphasia. There are lost (1) voluntary speech; (2) the repetition of speech; (3) the ability to read aloud. This is, of course, the purest form of motor aphasia. Interruption of the tract *Aa* gives rise to subcortical sensory aphasia. There are lost (1) the understanding of speech; (2) the repetition of speech; (3) the ability to write from dictation.

In actual pathology this theoretical classification with groupings of symptoms must sometimes be modified on account of a variety of conditions. The most important modification is that produced by the existence of possible lesions of other centres concerned in speech. Thus the share taken by the visual receptive and apperceptive centres is of great importance in all persons who have been taught to read. They necessarily are concerned also in the production of writing. It is not, however, possible to represent the mechanism of these functions by a diagram as we have represented auditory and motor speech, for it appears that impulses from the visual centres must pass through the receptive centre for speech, or *A*, before being transferred to the arm centre or the speech centre. The same is true for tactile impressions. These are of importance chiefly in blind persons who have been taught to read with their fingers, in whom, indeed, they may be equal in importance to the visual centres in normal persons. Various complicated diagrams have been devised for the purpose of exhibiting the influence of all these centres upon speech, and Mills has introduced an additional naming centre, situated in the third temporal convolution, in which perceptions are given the names that properly belong to them. A source of error is the fact that lesions may be only partially destructive, or may be so extensive as to involve two or more tracts or centres at the same time. Under these circumstances the symptoms become very complex, and it is often impossible to determine the extent of the physiological disturbance that had been produced. Usually, however, the localization of these lesions is not difficult, on account of the predominance of certain characteristic localizing symptoms.

Disturbances of writing, apart from disturbances of speech, may also occur. In *agraphia*, voluntary writing and the ability to copy are lost, while the ability to read is preserved. *Cortical alexia* is characterized by the loss of (1) the recognition of written words and (2) voluntary writing while speech may be intact. In *conduction agraphia*, voluntary writing and voluntary copying are lost; while the ability to read is preserved, that is to say, it corresponds exactly to the preceding form. Under such circumstances paraphasia may exist in this

type. *Transcortical agraphia*: loss of voluntary writing, and ability to do mechanical copying and writing. *Transcortical alexia*: loss of the ability to read, with preservation of the power of voluntary writing and of copying. There may be interruption of the tracts to the periphery giving rise to subcortical agraphia, in which voluntary writing and copying are impossible, but the power of reading is preserved. *Paragraphia* never occurs in this form. *Subcortical alexia* is inability to read and copy; voluntary writing is preserved. All of these forms may coexist with the various types of aphasia. In testing the patient for alexia, the following symptoms should be examined: (1) Voluntary writing; (2) writing from dictation; (3) copying; and (4) the recognition of letters either spoken or written. In testing patients for voluntary writing with the left hand, it must be remembered that many aphasics give mirror writing. The following terms also are used in connection with aphasia: *aphrasia*, the inability to form sentences with words; *dysphrasia*, the imperfect formation of sentences; *apraxia*, the total loss of speech.

Anarthria.—By anarthria is meant a disturbance of the peripheral motor mechanism of speech resulting from disease of the nuclei in the medulla or of the peripheral nerves arising from them. This may vary in degree from complete aphonia, or loss of power to make sounds and words, which occurs in bulbar paralysis, or the aphonia of laryngeal paralysis, in which whispering speech is still preserved, to mere inability to pronounce correctly certain consonants as a result of local paralysis or paresis of the lips and tongue. Anarthria may be permanent or temporary; or in cases of slight paresis, recurrent, giving rise to intermittent claudication of speech. It is tested by directing the patient to repeat letters of the alphabet, to count, or to repeat words with long syllables, and difficult consonants, as "artillery," "extraordinarily," etc. Allied to anarthria, but perhaps the result of certain functional disturbances, are *stuttering* and *stammering*. In the former, if the patient attempts to speak, there is inhibition of motion for a longer or shorter interval, and then the word may be pronounced with explosive violence, and the following words of the sentence spoken normally. In *stammering* there is frequent repetition of the first two or three consonants of the word, particularly if these happen to be labials. Stuttering and stammering are associated sometimes with defective intelligence. Finally, there is a series of disturbances of speech in which intellectual derangement is apparently the chief factor. These may perhaps be forms of aphasia due to partial destruction of the concept centre or centres. Among them may be mentioned the inability or unwillingness to speak that occurs in the *mutism* of the insane, in hysteria, and after a severe illness, as scarlet fever, causing cerebral fatigue; tendency to excessive speech, *logorrhea*; the omission of syllables, with interruption of the speech and tremulousness of the tongue and lips, which is particularly characteristic of general paresis, difficult words, such as those mentioned

above, being pronounced imperfectly, as "arly" for artillery," or even less accurately. *Scanning* speech, in which the words are separated by considerable intervals, and are spoken with a peculiar drawl and a descending cadence, is particularly characteristic of multiple sclerosis, but may occasionally occur in general paresis and in hereditary ataxia. Other forms are *explosive*, or *staccato* speech; and a peculiar, slow, drawling utterance occasionally termed *bradylalia*, which occurs in certain states of mental depression. *Echolalia* occurs almost exclusively in imbeciles, and is characterized by the repetition of all sounds heard.

SECTION III

OBJECTIVE DIAGNOSIS. DATA OBTAINED BY OBSERVATION

CHAPTER IX

FIRST SIGHT IMPRESSIONS AND GENERAL EXAMINATION OF THE EXTERIOR

FIRST SIGHT IMPRESSIONS

THE first sight, striking impression, is always to be noted. "Very sick," "comatose," "collapsed," etc., or "robust," "cyanosed," etc., are important memoranda. To the experienced practitioner, the opinion formed at first glance is often of great diagnostic significance. The general appearance of the patient may afford much information.

Location of Disease.—A general survey will often indicate which system is the probable seat of the disease. For instance, violent respiratory action points to the lungs; paralysis, to the nervous system; an enlarged abdomen, to disease of the viscera of that region.

Impairment of consciousness and convulsions are readily recognized. The two often go hand in hand, but in some instances, as in fainting fits, consciousness is not lost. The following list includes the most striking forms of general abnormal vital conditions which are recognized at first sight.

I. Unconsciousness.—(a) **Somnolence.**—This is a condition in which consciousness is lost, but regained upon any slight stimulation.

(b) **Stupor.**—This is a condition of unconsciousness from which the patient is aroused with difficulty. These two conditions may occur in any of the conditions in which coma may develop when the causative factor is not overwhelming.

(c) **Coma.**—This is a condition of unconsciousness, with suspension of perception and volition. Sensation and motion, however, are not always abolished. Coma always is of grave significance, indicating a serious disturbance of the cerebral functions; it occurs in a number of conditions, and presents different symptoms according to the cause; it may arise from (1) injury to the head, (2) disease of the brain or spinal cord, (3) the action of exogenous or (4) endogenous poisons.

1. INJURIES TO THE HEAD.—These injuries may produce laceration of the brain substance, with cerebral or meningeal hemorrhage or cerebral concussion. Coma resulting from such an injury is usually recognized by the history and the visible signs of a wound. A head injury in a comatose person, however, may be the result of a fall due to a sudden onset of unconsciousness.

2. DISEASES OF THE BRAIN AND SPINAL CORD.—Coma may be due to hemorrhage into the brain (apoplexy); to epilepsy (postconvulsive); to cerebral embolism or thrombosis; to thrombosis of the cerebral sinuses; to cerebral abscess; to pachymeningitis, leptomeningitis, or epidemic cerebrospinal meningitis; to cerebral syphilis; to general paralysis; or to multiple sclerosis. In a given case of coma the etiological diagnosis is based on the history if it is obtainable, and on the symptoms, which are fully described in the special part, in the sections devoted to the different diseases.

3. THE ACTION OF EXOGENOUS POISONS.—Coma may be produced by the influence of certain poisons: alcohol, opium, chloral, cannabis indica, the bromides and other narcotics, when taken in toxic doses.

Alcohol.—The coma of acute alcohol poisoning is profound, and is accompanied by great muscular relaxation. In the early stage the vessels of the face and neck are engorged, and the skin is moist and warm; later, the face is pale and ghastly, and the skin becomes cold. The pupils are usually moderately dilated and do not react. The pulse is rapid, strong at first, then more and more feeble. The respiration is stertorous and heavy. The sphincters usually are not relaxed. The body temperature is subnormal.

Opium.—The patient falls into a deep sleep, from which he usually can be aroused momentarily by loud shouting or violent shaking, and which is succeeded by the profound coma of opium-poisoning. The face is red and often cyanotic at first, later becoming pale and livid. The pupils are contracted to the size of a pin-point. The skin is dry until the approach of death. The breathing is slow, deep at first, then very shallow.

Chloral.—The coma of chloral poisoning follows the deep sleep caused by the drug. It is profound, and the patient cannot be aroused. Absolute muscular relaxation exists. The face is white and livid; the skin is covered with a cold sweat. The pupils at first are contracted, but soon become widely dilated. The pulse, while perhaps a little slowed at first, soon becomes rapid and thready, finally being imperceptible. The respirations are slow and labored at first, and then are shallow and feeble.

Infections.—Pernicious malaria, at the onset, and other severe infections late in the course of the disease, may cause coma, the result of overwhelming exogenous intoxication. The coma of the infectious fevers is produced gradually, and ordinarily is preceded by insomnia, by delirium or by other signs of cerebral disturbances. The condi-

tion of muttering unconsciousness with apparent wakefulness in these conditions is known as *coma vigil*.

4. **THE ACTION ON ENDOGENOUS POISONS.**—Coma due to endogenous toxins may occur in the course of Bright's disease, acute yellow atrophy, diabetes, Addison's disease, heat stroke, etc. The symptomatology of these conditions is discussed under the respective heads.

II. **Syncope.**—The attack, which may be preceded for a few moments by yawning, sighing, or nausea, is sudden and short. The face is pale and calm, the pulse feeble and imperceptible, the extremities cold. The breathing is quiet during the attack. The pupils are dilated and respond to light. There is no pain. The condition is the result of acute cerebral anemia, and occurs as a result of sudden emotion (depressing) or follows hemorrhage and shock.

III. **Collapse or Shock.**—The symptoms are those of prostration, with partial loss of consciousness, or the mind may be perfectly clear. The face is pale, pinched, and bathed in perspiration. The skin is wrinkled and cool and clammy. The hands are cold. The eyes are sunken and encircled by dark rings. The voice is weak and suppressed. The pulse is rapid and thready, and the blood pressure low. The temperature falls below the normal. The respiration may be hurried or shallow, sighing, and gasping. The urine is scanty or may be suppressed.

Collapse may be due to severe pain from any cause; to trauma; to operative insult; to hemorrhage, external or internal; to perforation of abdominal viscera; to peritonitis; to excessive watery discharge, as in cholera or serous purging. It may be due to pernicious malarial fever, and is then attended by coma. It may mark the termination of an infection, notably pneumonia. Sudden pneumothorax, embolism of the pulmonary artery, or rupture of a pulmonary abscess is attended by collapse. It may be present in the terminal stage of ulcerative endocarditis, and is not unusual in acute pancreatitis.

IV. **Convulsions.**—By convulsion is meant a series of involuntary contractions of the muscles of the limbs, face, and trunk, associated usually with unconsciousness. They may be *tonic*, that is, a continuous contraction of the affected muscles, or *clonic*, rapidly repeated contractions. Spasms is a term applied to local contractions of a part. Convulsions may be classified as follows:

1. **Convulsions with Loss of Consciousness.**

- (a) Epilepsy, "*haut-mal*."
- (b) Infantile convulsions. Usually reflex from indigestion.
- (c) Uremia. The coma alternates with convulsions.
- (d) Diabetes.
- (e) Alcoholism and sunstroke.
- (f) Hysteria.
- (g) Organic brain disease (syphilis, tumor, softening, etc.).

2. **Convulsions without or with only Partial Loss of Consciousness.**—Hystero-epilepsy, focal or Jacksonian epilepsy, cerebral embolism,

thrombosis or hemorrhage, spasms of various kinds, tetanus, hydrophobia, and strychnine poisoning.

3. **Convulsions with Vertiginous Movements.**—The forms of vertigo are gastric, aural, and labyrinthine (Ménière's, also paroxysmal), ocular, cerebellar, from congestion of the brain (reflex), epileptic.

V. **Vertigo.**—In vertigo the patient feels as if his head were swimming; or as if he were whirling around, or were falling forward or backward—subjective vertigo; or as if all surrounding objects were whirling around him or rising up toward him or pulling away from him while he himself remains stationary—objective vertigo. Vertigo is a symptom of many disorders, and usually accompanies any disturbance of the circulation within the cranium. It may occur in diseases of the heart, liver, kidneys, stomach, bloodvessels—arteriosclerosis, cerebral hemorrhage or apoplexy, cerebral embolism and thrombosis—blood, nerves, brain, spinal cord, ear, and eye; in disturbance of equilibrium; after the taking of certain drugs; and after applications of electricity, as galvanization of the skull. The presence of a foreign body in the ear, the insufflation of air into the Eustachian tube, and disease of the labyrinth and of the auditory nerve may give rise to vertigo. Ménière described a disease associated with paroxysmal vertigo. Paralysis of the ocular muscles, eye-strain, and astigmatism may be accompanied by vertigo. Laryngeal vertigo is often closely associated with epilepsy. The larynx is usually normal in this affection, but in one case it contained a polyp. Tickling or burning in the larynx usually precedes the vertigo. Toxic vertigo occurs after the ingestion of large doses of quinine or salicylates, and after excessive indulgence in alcohol, tobacco, coffee, tea, or opium. The vertigo which develops as a sequence of infectious diseases probably results from the action of the toxins. This is the case in the vertigo of malaria and in the paroxysmal paralyzing vertigo of Gerlier.

Functional vertigo includes that which occurs in seasickness and mountain sickness, the dizziness felt when looking down from a height or at running water, and on jolting, swinging, or revolving. The diagnosis of the cause of the vertigo can only be made after an examination of the patient and by exclusion.

GENERAL EXAMINATION OF THE EXTERIOR

The Personal Appearance.—From the general appearance of the patient, the observant diagnostician may frequently garner facts that may be a very real aid in arriving at a diagnosis.

The Apparent Age.—The apparent age of the patient should be estimated from his appearance, and compared with the actual age when this is learned later. In this way the physician will be able to judge whether the patient is aging too rapidly or bearing his age well. Gray hair in a person under thirty-five generally indicates a feeble constitution and premature age. The presence of wrinkles at the

corners of the eyes and of "crows'-feet," and of dull, dry, lusterless eyebrows, should be noted as indicating aging, whether the person has lived long or not. In women approaching forty, who do not gain in flesh, there is often a suggestive prominence of the angles of the jaw and sternomastoid muscles, with a certain loss of roundness and elasticity of the cheeks. The latter appearance, however, may be due to the loss of the molar teeth.

The Temperament and Constitution of the Patient.—By diathesis is meant the inherited state or condition of the body which seems to predispose to certain diseases. Five varieties of diathesis are described: the gouty, the strumous, the nervous, the bilious, and the lymphatic. While groups of individuals may be classified under one of these diatheses, it is well not to lay too much stress upon them for diagnostic purposes.

In the *gouty* (sanguine, lithemic) diathesis the osseous system and muscles are well-developed, the nutrition active, and the patient usually robust in appearance. The digestion is good, the respirations are deep, the circulation is well carried on (as shown by the florid skin and the large heart), the pulse is firm and steady, and the pressure in the arteries is high. The head is large and the jaw prominent; the teeth are good. The individual with this diathesis is predisposed to the cardiovascular changes of advancing age.

In the *strumous* (tuberculous, phthisical) diathesis the appearance of the face is rather coarse; the bones of the chest are small; the long bones are slender, while their epiphyses are large; the forehead is broad and prominent, the lips full, the alæ nasi thick, the teeth carious, the lower jaw light and thin, the hair fine and often of a light hue, the eyelashes long, the eyebrows arched and often heavy and the chest flat. In this diathesis the nutrition is poor so that inflammations are usually sluggish; diseases of the bones, and forms of tuberculosis are apt to be more severe.

In the *nervous* (neuropathic) diathesis we see small, active, restless beings, with small bones and large muscles. They are full of energy, and carry on large business operations or other ambitious undertakings. The features are well-formed and the eyes active. Such types readily become the victims of overwork, dyspepsia, and early breaking down of the nervous system. They possess idiosyncrasies toward drugs, particularly opiates.

In persons of the *bilious* diathesis we find a dark skin, dark hair, muddy conjunctiva. They are usually not well nourished. Their digestion is poor and they are subject to attacks of so-called biliousness. Sick headaches are common. Fatigue is not borne well.

In the *lymphatic* diathesis there are lack of energy and sluggishness; such persons are unable to keep up in the wear and tear of life. They are usually pallid and have soft muscles.

Cachexia.—This is also to be noted. It arises from the ravages of disease, especially when the number of the red cells of the blood

is reduced and the hemoglobin diminished. Cachexia is caused especially by syphilis, cancer, and chronic malarial poisoning. In cancer of some parts of the digestive apparatus—and, indeed, in all forms of chronic disease of the digestive tract—a cachexia develops. The anemia from poisoning with lead, arsenic, and other metallic poisons produces an appearance to which the term cachexia has also been applied, although in truth it only resembles that state.

Stigmata of Degeneracy.—As a result of the careful studies of Lombroso and others we have learned that certain alterations in the structure of the body are frequently associated with disturbances in the mentality of the individual, or the capacity of resistance and assimilation of the individual tissues. Naturally the significance of these stigmata varies considerably in different individuals, and they should be regarded as suggestive rather than decisive.

Anomalies in the general growth include dwarfism and giantism. Dwarfism may be of various types, the most important being the true dwarf, in whom all portions of the body are small; the rachitic dwarf, in whom distinct anomalies in the skeleton occur; the cretin or dwarf of congenital myxedema. Giants also may be of two types: the true giant and the acromegalic giant. Most of these varieties are really types of disease. The modification of growth may affect only certain parts of the body. The head may be abnormally large or small without any distinct evidence of disease. A more characteristic stigma is excessive elongation of the arms, approximating the simian type, or imperfect development of the lower portion of the body; small thin legs associated with a well-developed torso. Not infrequently we notice in otherwise well-developed persons that the chest is abnormally narrow—that is, the thorax as a whole is poorly developed. This may be associated with the congenital hypoplasia of the cardiovascular system described by Virchow. The more pronounced forms of degeneracy cannot but affect the health of the individual.

Other stigmata of degeneracy consist of alterations of the ears: absence of the lobe, absence of the antihelix; position of the external ear with relation to the head, so that it stands out almost perpendicularly; presence of a small point upon the helix, a vestige of the animal type; adherence of the lobe to the side of the head.

Abnormalities of the eyes: they may be unusually close together or far apart; placed at different levels; they may present various changes in the ocular muscles. Even the growing together of the eyebrows is looked upon as a slight stigma of degeneracy.

Excessive breadth of the nose, imperfect closure of the nostrils, marked deviation of the septum, all these anomalies are included in this category. In the mouth: various irregularities, such as wide separation of the teeth; abnormal development of the canines; the prognathic or agnathic jaw; high arching of the palate; cleft palate—are all found more frequently among persons otherwise degenerate than in normal individuals.

In the hands: supernumerary digits, exaggerated webbing of the fingers, or the so-called cleft hand, that is, separation of two of the metacarpal bones, have been noted. Similar anomalies have been described in the feet. Hypospadias and epispadias, abnormally small size of the external genitalia, are supposed to be quite characteristic features. Anomalies of the hair consist in abnormal growth upon the forehead, upon the back, following the course of the spine, or excessive irregularities of the hairy growth on other parts of the body. Delayed puberty, or the persistence of the infantile, or development of the contrasexual type—that is, the feminine type in men, and the masculine type in women, are also examples of degenerate tendencies.

The Attitude and Gait of the Patient.—The attitude of the patient gives information as to his physical vigor, and, to a certain extent, of his alertness of mind.

Decubitus.—The attitude of the patient in bed is often significant. He may assume the *active dorsal*, or the *side position*, with the body arranged so that it is comfortable and unconstrained. Then slight indisposition only is present. On the other hand, the *side position*, the *dorsal position*, or the *upright* or *semi-upright* position may be assumed.

Side Position.—A patient with acute pleurisy or pneumonia will lie on the affected side, so as to limit its motion as much as possible. If effusions are present, by lying on the side of the effusion, pressure is removed from the heart and the unaffected lung—an obvious advantage. At times, in case of thoracic aneurysm, or of movable thoracic tumors, if situated on one side, the patient will lie on the side which is the seat of the disease.

Dorsal Position.—When the position is assumed in grave disease it is called *passive dorsal*, because it is often assumed without volition of the patient. In grave cases of typhoid or other low fevers the patient lies upon the back and shows a marked tendency to slip down in the bed. A healthy baby a few months old finds motion an almost ceaseless delight; in rachitis, on the contrary, the little patient lies as quiet as possible, even refraining from crying, because all motion is painful. In cerebrospinal meningitis the head is drawn backward and downward, and the muscles at the back of the neck are rigidly contracted.

In acute disease involving the peritoneum or neighboring organs, such as acute general peritonitis, appendicitis, or salpingitis, the patient lies on the back with the legs flexed upon the thighs and the thighs upon the abdomen. Motion is avoided as much as possible.

Lateral Position.—The lateral or dorsal position, with legs drawn up and trunk and head bent over to meet them, is assumed in hepatic and intestinal colic and during the throes of labor.

The Semi-upright or Upright Sitting Position.—In an acute attack of asthma the patient is found sitting up in bed, or in a chair, possibly by an open window. The patient sits with the chin raised and the

head erect, the hands grasping the arms of a chair or the bedclothing, so that, by fixing the chest, the accessory muscles of respiration can be of the greatest assistance in supplementing the diaphragm. In emphysema, in its late stages, or when complicated with bronchitis and asthma, the same position is assumed almost constantly.

In pericarditis with effusion, in large pleural effusions, and in advanced heart disease with anasarca the patient is unable to lie down on account of the smothering feeling which the recumbent position induces. In diaphragmatic pleurisy the position assumed is very characteristic—the erect sitting posture, with the body leaning forward and laterally, to relieve the pain.

The Prone Position.—The patient is found lying upon the abdomen. It gives relief to abdominal pain or to colic of any form. The pain of an ulcer of the stomach, or aneurysm, or of caries of the vertebræ may be thus mitigated.

Unclassified Positions.—Irregular or bizarre positions are usually assumed in affections of the nervous system, particularly in hysteria.

In tetanus opisthotonos occurs. The body is supported on the head and heels and the trunk is arched upward, because of tonic contraction of the spinal muscles. In strychnine poisoning with tonic convulsions, the same position may be assumed. Emprosthotonos, the converse position, in which the body rests on the forehead and feet, is occasionally assumed in tetanus and also in strychnine poisoning.

Restlessness.—This may occur on account of pain, or because of irritation or anemia of the nerve centres. In cases of moderate cerebral hemorrhage and of shock there is great restlessness. In profuse hemorrhage the resulting cerebral anemia manifests itself in restlessness, with sighing and gasping. In chorea there is more than restlessness—there is constant twitching of muscles with jerking from one side of the body to the other. It is a remarkable symptom of the active period of exophthalmic goitre. In cerebral meningitis the patient tosses from side to side or lies with the head retracted and pressed deeply into the pillow. In hysterical convulsions the patient tosses wildly to and fro, screaming, laughing, or crying; or coma may be mimicked.

Gait.—An irregular gait, a limp or hobble, may be due to a painful, weakened, or injured limb. The gait is sometimes exceedingly characteristic in nervous diseases. The hemiplegic patient advances the sound limb, and then brings the other up to it by lifting the pelvis and swinging the paralyzed limb around by a movement of circumduction.

In *locomotor ataxia* there is uncertainty in the gait, which may be felt only by the patient or may be apparent also to the observer. There is irregularity in the line of progression, or the movements become very jerky and erratic. As there is very little motion at the knee, because it is spasmodically braced, the pelvis is slightly tilted until the foot is released; the foot is then raised unnecessarily high, jerked rapidly forward and outward, and brought down with a sudden stamp, or flail-like action, on the heel. The patient's centre of gravity under-

goes several changes at each step, so that he swings from side to side. He cannot walk in the dark, and at a later stage requires the aid of canes to prevent him from falling forward. The so-called steppage gait is somewhat akin to the gait of ataxia except the toes are dragged as a result of the foot-drop that is present from the accompanying *peripheral neuritis* of the external popliteal nerve.

FIG. 8



Spastic paraplegia, cross-legged progression. (Dereum.)

FIG. 9



Hysterical astasia-abasia. (Lloyd.)

In *paralysis agitans* the attitude and gait of the patient are peculiar. The head and body are thrown forward and fixed in that position; the arms are slightly abducted and partly flexed, the hands being in the position in which a pen is held or a pill rolled. The legs are also bent at the knees. The gait is festinating—that is to say, each step becomes more rapid than the preceding, until the patient is prevented from falling only by catching hold of something.

In *spastic paraplegia* the patient walks with two sticks. He leans on the left one, arches the back, and then lifts the pelvis and the right limb as far from the ground as possible, but cannot quite clear it. The leg is rigid and the foot dragged around in a semicircle. The toe has a marked tendency to stick to the ground, and is brought forward

with a sweeping sound. The knees have a tendency to interlock, and the foot that is brought forward is apt to cross in front of the other.

In *disseminated sclerosis* the gait is somewhat jerky and resembles the gait of ataxia or of tumor of the cerebellum.

Cross-legged Progression.—This form of gait is seen in children with spastic paraplegia, and occurs because of contracture in the calf muscles. When the child begins to walk, one foot gets over in front of the other. Sometimes a swinging oscillation of the body occurs, which may persist throughout adult life.

The gait of *pseudohypertrophic muscular paralysis* is known as the waddling gait. This oscillating character is assumed in order that the body be so inclined "as to bring the centre of gravity over each foot on which the patient successively throws his weight, because the weak gluteus medius cannot counteract the inclination toward the leg that is off the ground unless the balance is exact" (Gowers). The position assumed in getting up from the floor, as described by Gowers, is pathognomonic. The patient turns over in the all-fours position, raises the trunk with his arms, rests the trunk upon the extended hands, then extends the knees, pushes back with the hands until he can grasp one knee with the corresponding hand, then grasps the other knee, and pushes up the trunk by gradually raising the point of support for the hand upon the thigh.

The swaying gait, like that of a drunken man, is significant of cerebellar disease. (See Station.)

The gait of paramyoclonus multiplex and that of Thomsen's disease are also peculiar. (See Muscles.)

Station.—*ASTASIA AND ABASIA.*—These terms are employed to define the loss of power of standing and of walking respectively, without paralysis. Hysteria is the most common cause.

Ataxic Astasia in Tabes Dorsalis.—The inability to stand is observed either with (1) the eyes closed, or (2) with the eyes open and the toes and heels in contact, or (3) with the eyes open and the feet apart. The latter occurs in the highest degree of ataxia, and may be followed later by complete loss of power of standing.

Swaying.—If a healthy person stands with the eyes shut and the feet close together, the body will sway slightly. In a patient with locomotor ataxia this swaying is greatly exaggerated.

In pseudohypertrophic paralysis, when the patient stands, there is that extreme curvature of the spine known as lordosis. In the later stages of this affection there is posterior or lateral convexity of the spine with astasia. In paroxysms of Ménière's disease loss of the power of standing may be absolute. The patient may be hurled to the ground and be quite unable to rise or sit up. In disease of the middle lobe of the cerebellum swaying from side to side in wide oscillations is observed. The appearance is like that of a drunken person. While the walk is peculiar, the patient can usually sit up.

The Nutrition.—The nutrition of the body is estimated by the color of the skin, the amount of subcutaneous fat, the degree of muscularity,

and the size and shape of the osseous system. Hence, we estimate the degree of physical development of the individual by the size and weight, by the nutrition of the muscles, and by the state of other tissues. The recognition of malnutrition, as shown in lack of muscle tone and diminution of weight, is often sufficient to point the way to successful treatment by hygienic methods.

Size.—Such expressions as stout, spare, slender, thin, tall, and short are used to denote variations in size from the recognized normal standard. "Stout" usually expresses an increase in girth and a moderate excess of flesh over the normal. In some cases, especially in women, stoutness is used as an euphemism for corpulency, but not often for that excess of fat properly called obesity. A tendency to take on flesh after the age of forty-five, especially if the person's occupation is sedentary and his habit of body inactive, is not to be regarded as favorable. Persons who are tall and thin, with a long, narrow thorax, especially if they have become tall rapidly after puberty, are commonly looked upon as delicate, and as especially liable to tuberculosis in their earlier years of life.

Some patients have an appearance which is well-described and understood by the word spare. The figure is compactly put together, with small bones and a scanty allowance of fat, and there is a tendency to leanness rather to roundness of form.

In still others muscle and bone predominate, and the figure is apt to be angular, as in those described as wiry. They are often possessed of great muscular power and resistance to strain and disease.

In estimating the patient's size and weight, it is important to ascertain if he has regular habit of taking on flesh at certain periods of the year, or if the obesity has developed suddenly or followed acute disease.

Weight.—The weight affords a precise estimation of the size. While the eye can estimate approximately the weight of the body and the degree of emaciation, the physician should make it a rule to ascertain the weight accurately by means of scales. The relation of body weight to height is of importance. It is also important to know the average weight of the individual at different periods of life.

Hutchinson's table enables us to judge the average weight of a healthy man of a given height:

A man of 4 ft.	6 in. to 5 ft.	0 in.,	ought to weigh about	92.26 pounds
A man of 5 ft.	0 in. to 5 ft.	1 in.,	ought to weigh about	115.52 pounds
A man of 5 ft.	2 in. to 5 ft.	3 in.,	ought to weigh about	127.86 pounds
A man of 5 ft.	4 in. to 5 ft.	5 in.,	ought to weigh about	139.17 pounds
A man of 5 ft.	6 in. to 5 ft.	7 in.,	ought to weigh about	144.29 pounds
A man of 5 ft.	8 in. to 5 ft.	9 in.,	ought to weigh about	157.76 pounds
A man of 5 ft.	10 in. to 5 ft.	11 in.,	ought to weigh about	170.86 pounds
A man of 5 ft.	11 in. to 6 ft.	0 in.,	ought to weigh about	177.25 pounds

Loss of Weight.—The question of weight is an important one in disease. In the course of wasting diseases we learn the effects of treatment, or, on the other hand, the march of disease in spite of treatment. In obscure cases, as in suspected tuberculosis, persistent loss of flesh

is a grave diagnostic and prognostic sign. After acute disease, if the patient is weighed every week, the onset of insidious sequelæ, as tuberculosis, may be detected. Flesh is lost in almost all diseases, acute or chronic; but it becomes of special diagnostic moment in the latter. It is most noticeable in tuberculosis, cancer, cirrhosis of the liver and kidneys, diabetes, in anemias, and in cachectic conditions due to prolonged suppuration or chronic diarrhea, especially amebic dysentery, in gastric neurasthenia and anorexia nervosa. If emaciation is present, ascertain its degree, its rate of development, and its possible relation to unusual mental care or to acute disease. Slow progressive emaciation is of serious moment. Again, while loss of weight attends all diseases of the digestive tract which interfere seriously with nutrition, it progresses more rapidly and steadily and attains a greater degree in malignant disease than in functional or other diseases.

False Increase of Weight.—In certain cases of great anasarca, in malignant diseases of the abdomen, especially huge cysts of the ovary in women and sarcoma of the kidney in children, there may be actual increase of weight due to the accumulation of fluid or to the new growth, though the rest of the body is manifestly emaciated.

Weight in Children.—In babies and children fat is more likely to be a sign of good health than in adults. Nevertheless the quality of the flesh is to be taken into consideration. There are fat and flabby babies and children, and there are others who are fat but whose flesh has a firm solid feel. The former often gain and lose flesh rapidly, and when ill do not appear to have much resisting power. In fact, by the weight alone can we judge of the normal growth of the child.

Muscular Development.—Palpation shows whether the muscles are flabby or relaxed and large or small, firm or elastic. General lack of muscular development or muscular weakness is an important sign of malnutrition and may explain the nature of many symptoms. The muscular weakness can be approximately estimated by the degree of firmness of the muscle. Weakness of the muscles of the spine, with resulting curvature or inability to keep the erect posture, is sufficient cause for the occurrence of neuralgic pains in the course of related nerve trunks and for the displacement of organs within the thorax or abdomen, often causing functional disturbance. Forms of indigestion, from sluggishness of secretions, particularly of the bowels, follow in the wake of muscular debility and pass away as the muscles regain their tone. Moreover, weakness of the abdominal walls and separation of the recti muscles or diastasis favor dropping of the liver, stomach, and other organs, causing visceroptosis with its train of symptoms. Atrophy of muscles occurs because of disuse, because of sedentary occupation, or of a life of ease and luxury, with excessive feeding.

Increase in Weight and Size.—Increase in weight and size then may be due to changes in (1) the skeleton; (2) the muscles; (3) the adipose tissue; (4) the subcutaneous connective tissue as a result of accumulations of pus, blood, or serum; (5) to change in size of internal organs; (6) to new growths of or fluid accumulations in the body.

CHAPTER X

EXAMINATION OF THE FACE, HEAD, AND NECK

THE FACE AND ITS EXPRESSION

THE face is a mirror in which are reflected all degrees of ill health from that which amounts only to temporary indisposition and depression up to the gravest cachexia. Moreover, the face tells of the presence or absence of pain, and to a certain extent of its character.

The late Austin Flint, Sr., tersely described the various appearances of the face in disease, with their clinical significance, as follows:

The Facies of Renal Disease.—In some cases of acute albuminuria and of chronic parenchymatous nephritis, puffiness of the face from edema, with notable pallor, renders the aspect highly diagnostic.

The Malarial Facies.—Pallor of the face, sallowness, and slight puffiness, if renal disease is excluded, point to malarial disease.

The Facies of Carcinoma.—Notable anemia, a waxy or straw-colored complexion, and more or less emaciation, in combination, render the aspect marked in some cases of malignant disease.

The Typhoid Facies.—In the middle and later periods of typhoid fever the countenance is often dull, besotted, expressionless. This facies may be present in the typhoid state, which is incident to disease other than typhoid—e. g., pneumonia.

The Facies of Acute Peritonitis.—The upper lip raised so as to expose the front teeth gives an aspect which characterizes, in a certain proportion of cases, acute peritonitis.

The Facies of Acute Pneumonia and Hectic Fever.—Circumscribed redness of one or both of the cheeks, with abruptly defined borders, is diagnostic of acute pneumonia. If it is observed in a case of chronic pulmonary disease, it denotes the so-called hectic fever, and is a sign of phthisis.

The Facies of Exophthalmic Goitre.—Projection of the eyeballs, giving to the face a remarkably staring and sometimes ferocious expression, conjoined with enlargement of the thyroid body and frequency of the pulse, is distinctive of the affection known as exophthalmic goitre—Graves' or Basedow's disease.

The Choleraic Facies.—In the collapse stage of cholera the face is contracted, sometimes wrinkled; the cheeks are hollow, the eyes sunken, the skin livid, and the expression denotes indifference. This combination is, however, to a certain extent combined in the state of collapse which occurs in some cases of pernicious intermittent fever and in other pathological connections.

The Hippocratic Facies.—This facies denotes the moribund state. The skin is pale, with a leaden or livid hue; the eyes are sunken, the eyelids separated, and the cornea loses its transparency; the nose is pinched and the eyes are retracted; the temples are hollow and the lower jaw drops.

The Facies in Children.—Inspection is even more important in the case of children than in adults. The pale, pinched, weazened face of some babies who have snuffles, ulcers, or striated lines at the corners of the mouth, and look prematurely aged, with prominent forehead and a depressed nasal bridge and retroussé tip, characterizes inherited syphilis.

The head is unusually large, with flattened vertex, projecting forehead, and open fontanelle in rachitis. The head becomes very much enlarged, the eyes prominent, the bones of the face remaining small, the expression vacant in hydrocephalus. The dull apathetic expression, with thickened lips, the small nasal orifices, and the gaping mouth, are characteristic of adenoid disease of the pharynx with tonsillar hypertrophy.

The thickened lips, the protruded tongue, with saliva dribbling from the open mouth, the flattened nose, with the idiotic expression and pallid waxy skin, are easily recognized in cretins.

The red swollen face, the reddened, weeping eyes, and running nose make a very striking picture in measles.

An irritating, excoriating discharge from the nose in a child may indicate the existence of nasal diphtheria.

The Facies in Nervous Disease.—All varieties of mental aberration are reflected in the face. The suspicious, the revengeful look; the wild look; the plaintive, depressed; the vacant, listless, peaceable look; all come to be recognized very readily by those who see much of the insane. In hysteria, expressions of varied emotions are seen; in neurasthenia, a worn and wearied aspect of countenance is noticeable.

Spasm and Tremor.—Change in the expression and appearance of the face very frequently occurs because of change in the function and nutrition of the muscles, on account of central or peripheral disease of the nervous system. On this account we have facial spasm or tremor in convulsive tic, chorea, epilepsy, tetanus, hysteria, or as a result of habit, and unilateral, bilateral, or local facial paralysis.

In *peripheral facial palsy* the paralyzed side of the face has a staring, vacant expression, owing to the fact that the eyelid is motionless. The angle of the mouth on the affected side is depressed. The whole paralyzed side is devoid of wrinkles, and has a smoothed-out, glazed appearance; the contrast with the normal side is most marked when the patient smiles or frowns (Fig. 10).

In *glossolabial palsy* there is progressive paralysis with tremulousness of tongue and lips; progressive failure of articulation, and dribbling of saliva. Sometimes the patient is able to open the lips, but unable to close them without the aid of the fingers. The mask-like expression

of immobility in *paralysis agitans* has been described as Parkinson's mask.

Facial hemiatrophy is a peculiar affection, characterized by progressive wasting of the bones and soft tissues of one side of the face. The disease is rare; it begins, as a rule, in childhood, but may develop in later life. The patient looks as if the face were made up of two halves from different persons. It must not be mistaken for the facial asymmetry which is associated with congenital wry neck, and is distinguished by contraction of the sternomastoid muscle from birth (Fig. 11).

FIG. 10



Complete facial palsy. Patient unable to close eye of the affected side. (Dercum.)

FIG. 11



Facial hemiatrophy. (Lyman.)

Color of the Face.—A full account of the color is given in the chapter devoted to the Appearance of the Skin.

Contour of the Face and Head.—The changes in contour in *acromegaly*, *rachitis*, and *osteitis deformans* are described in the chapter on Bones and Joints. In *leprosy* the characteristic leonine countenance, *facies leontina*, is the result of the tuberculous outgrowths about the eyes and forehead. The outline in *myxedema*, *sporadic cretinism*, *hydrocephalus*, and *scleroderma* are described elsewhere.

Swellings of the Face.—The face is swollen and deformed in mumps, erysipelas, and smallpox, and to a moderate extent in measles.

The puffiness of the eyelids and general swelling of the face in *anasarca* due to Bright's disease and in *trichinosis* will be referred to in the chapter on Edema.

Of characteristic appearance is the swelling of the face in obstructive heart or lung diseases and in the spasmodic stage of whooping cough.

Temporary swellings may be due to urticaria or angioneurotic edema.

Local swellings occur as follows:

1. Of the *forehead*, in glanders, trichinosis, and thrombosis of the superior longitudinal sinus.
2. Of the *upper jaw*, in antrum disease and alveolar abscess.
3. Of the *lower jaw*, in alveolar abscess and actinomycosis.
4. In *front* of the ear, in mumps.
5. Over the *mastoid process*, in mastoiditis from ear disease and thrombosis of the lateral sinus.
6. In the *cheeks*, in gangrenous stomatitis and in obstruction of Steno's duct.

Anthrax and boils may cause swelling of any part of the face.

The Lips.—The lips are usually pale in anemia. They are livid in cyanosis from chronic lung or heart disease, with feeble circulation. Vesicles (herpes) are apt to appear upon the lips in common colds, in certain febrile diseases, particularly pneumonia, and with many women during or immediately following menstruation. An ulcer due to a chancre or epithelioma, and mucous patches may be situated on the lips.

A child with hereditary syphilis may show ugly fissures, or the scars which result from them, at the angle of the mouth.

In facial palsy the angle of the mouth on the paralyzed side is depressed and the skin is free from wrinkles. In glosso-labial-laryngeal palsy the lips tremble and twitch, and may have to be closed with the fingers after they have been opened. In general paralysis of the insane the lips tremble.

The Bloodvessels.—The veins are enlarged and tortuous in tumors of the neck, thrombosis of the lateral sinus, and in varicose aneurisms. The temporal arteries are prominent, rigid, and tortuous in atheroma.

THE HEAD

Abnormal Movement.—Irregular bizarre movements occur in *chorea*. A rhythmical nodding spasm of the head occurs in hysteria, in the epilepsy of childhood, or may be due to habit. Spasmodic jerking of the head with rotation to one shoulder, and elevation of the chin and rotation of the face to the opposite side, occurs in spasmodic torticollis.

Abnormal Fixation.—Spasm of muscles, myalgia, torticollis, arthritis deformans, scleroderma, and retropharyngeal abscess cause more or less fixation of the head. In tuberculous disease of the vertebræ the head is fixed and the pain is relieved by support. Fixation and retraction occur in meningitis, in tetanus, and in strychnine poisoning.

Enlargement.—Changes in the size and shape of the head are seen in *leontiasis ossea*, *rachitis*, *acromegaly*, *osteitis deformans*, which are discussed in the chapter on the Bones and Joints; in *myxedema* and *leprosy*; in *sporadic cretinism* and in *hydrocephalus*.

Hydrocephalus.—The enlargement of the skull is very conspicuous, and the disproportion of the cranium to the face is striking. The cranium

is rounded or globular in shape, and the fontanelles are seen to be very large, tense, and bulging; the sutures are widely separated. The disproportion in size between the face and head is apparently increased by the projection of the anterior portion of the skull. The axes of the eyes are directed downward, and they are partly covered by the eyelids because of the oblique direction of the orbital plates. The head is supported with difficulty. The eyeballs roll from side to side. Frequently there is strabismus. The skin is stretched tightly over the cranium and the hair is scanty. The veins are prominent (Fig. 12).

FIG. 12



Congenital hydrocephalus. Female, aged seventeen years. The thinness of the hair could not be represented. (Original.)

Diminution.—Diminution in the size of the head is seen in microcephaly (circumference less than seventeen inches). The head is usually abnormal in shape.

Sweats.—Sweating of the head is of common occurrence in rachitis.

The Hair.—The hair often indicates the state of the individual's nutrition. The abnormal growths and changes in the texture due to local parasitic disease will not be referred to here. Undue and rapid loss of hair in patches is indicative of syphilis. The hair can be easily

pulled out in large masses without causing pain. This falling of the hair must not be confounded with the excessive falling out which takes place during convalescence from acute disease, particularly typhoid fever, nor with that following an attack of gout or erysipelas. Loss of hair may follow severe neuralgias of the fifth nerve.

Color.—Early gray hair may go hand in hand with premature endarteritis. The term “canities” is applied to the diminished development of pigment. Premature gray color in well-defined patches occurs in nerve lesions, as paralysis of one of the branches of the fifth pair, and is a trophic change. Sudden change in the color of the hair, usually to gray, is said to occur at times under the influence of fright, mental anxiety, or deep emotion.

“Green” hair is seen in brass founders and workers in copper mines. “Blue” hair in laborers in cobalt mines and persons employed in the manufacture of indigo. Chemicals applied to the hair change its color—hydrogen peroxide bleaches the hair, pyrogallie acid turns it black.

The Fontanelles.—The fontanelles in a healthy child, with the exception of the anterior, close during the early weeks of life. The anterior fontanelle closes some time between the sixteenth and the twentieth month. New openings or fontanelles and loose bone plates, the normal fontanelles remaining open, are seen in so-called *craniotabes*—a condition found in congenital syphilis and rarely in rachitis.

Prominence or fulness of the fontanelles may be temporary or permanent. When the former, a passing fever with cerebral congestion may be the cause; when the latter, hydrocephalus or some other brain affection in which there is increase of intracranial pressure.

Depression of the fontanelles occurs in general atrophy, marasmus, and wasting diseases generally. In collapse it is of grave prognostic omen. In pneumonia and other respiratory affections with dyspnea retraction of the fontanelles is observed. The former affection, with cerebral symptoms, is thus distinguished from cerebral meningitis, in which the fontanelles bulge.

Delayed Closure.—In rachitis the fontanelles may remain open up to the third or fourth year of life.

The Bones.—The bones of the cranium may be thickened; they may be the seat of periostitis, necrosis, or caries. Nodes and painful doughy swellings, becoming indurated and due to periostitis, occur in syphilis. Necrosis and caries of the *frontal* bone are almost pathognomonic of syphilis. Necrosis of the jaw bone belongs to phosphorus poisoning. The mastoid process of the temporal bone is frequently affected in middle-ear disease.

Examination of the mastoid region should include the *occipito-atlantal articulation*. Disease of this articulation, and particularly tuberculous disease, causes stiffness of the neck or falling forward of the head.

Percussion.—McEwen, of Glasgow, has found that in cerebral abscess, tumor, and also in meningitis secondary to ear disease a higher pitched percussion note is heard over the affected area, and at the same time the percussion resistance is increased.

THE NECK

The position and movements of the larynx and trachea, the thyroid gland, the lymphatic glands (see Chapter XIV), and the vessels of the neck are to be observed. (See Chapter XX.)

The Larynx and Trachea.—These structures occupy the median line in health. They may be deflected to the right or left, and the deflection is more readily noticed at the lower part of the neck. Pseudodeflection may be due to atrophy of the muscles of one side. True deflection is due to an external growth or aneurism, or to disease within the thorax, as a thoracic aneurism or a mediastinal tumor. In chronic fibroid phthisis the trachea is often drawn over to the side of the affected lung.

Movements.—When the respiratory movement of the larynx and trachea is excessive and associated with dyspnea, the source of the dyspnea is of laryngeal origin. Tracheal tugging, usually determined by palpation, is particularly characteristic of aneurism of the descending portion of the aorta. (See Diseases of the Bloodvessels.)

Thyroid Gland.—Atrophy.—This is shown by absence of fulness, which would otherwise be present.

Enlargement.—Enlargement of the thyroid can be detected without much difficulty. It may be limited to one lobe or affect both lobes. The enlargement varies in size from a small localized swelling to large masses filling the median and lateral portions of the neck, pressing upon the trachea, and extending into the thorax. The swelling may be soft or hard to the touch. In the fibrous forms the swelling is not very large and is very much indurated. In the cystic forms of thyroid enlargement, fluctuation may often be detected; it may be localized to a small area of the lobe, or may be present over the entire affected lobe. In some cases a purring or thrill is transmitted to the fingers, synchronous with the heart's action and due to increased vascularity of the gland. Auscultation under these circumstances reveals a systolic murmur.

CHAPTER XI

EXAMINATION OF THE EYE AND EAR

THE EYE

INDIRECTLY the eye and the skin are the external structures that most frequently present evidence of disease in other organs. This is particularly true of the eye because it is a highly specialized organ, bearing close relationship to the nervous and vascular systems. Its close connection with the nervous system renders it sensitive to abnormal changes, and in diseases of this system the eye is therefore the one organ the examination of which is essential to make a diagnosis, and the converse holds true, that in the study of the changes in the eye reference must be made to the nervous system. In addition, diseases of the heart and kidneys, and certain systemic conditions, such as anemia, gout, rheumatism, diabetes, etc., often find expression in some eye change.

Through the ophthalmoscope we have unfolded to our gaze a living nerve head, the optic papilla, and the retinal vessels, which offer to our view the perfect cycle of the supply of an organ with arterial and the escape of its venous blood. We have here the only visible blood-vessels in the body and hence have the opportunity of observing and studying changes in the general circulatory system.

The Lids.—Edema.—This is a not infrequent symptom of renal disease (see Edema of the Face), and may occur in cases of profound anemia and chlorosis; after the prolonged use of arsenic, or in disease in the orbit or some of the periorbital sinuses and in general anasarca. The accumulation may take place during the night and become evident in the morning on rising. Morning puffiness is natural to some individuals, and, like swelling of the face following a debauch, is not to be confounded with edema.

Ptoxis.—Ptoxis, or drooping of the eyelid, may be congenital; more usually it is a symptom of disease within the brain. (See Paralysis of Third Nerve.)

Lagophthalmos.—Lagophthalmos, or imperfect closure of the lids, follows paralysis of the facial nerves. It may be due to a mechanical cause, as in exophthalmic goitre or orbital tumors.

Blepharospasm.—This, varying from an active closure of the lids from spasm to a slight twitching, is of a reflex nature, originated by excitation of a filament of the fifth nerve. It is always present to a greater or less degree in *photophobia*; this latter symptom is a frequent

associate of ocular disorders, and is found also in certain stages of meningitis, cerebral tumors, typhus, measles, etc. It accompanies many forms of headache, especially migraine, and may be the expression of a hyperesthesia of the retina in nervous subjects, apart from any actual inflammation of the membrane. Spasm of the orbicularis muscle is often seen in hysteria. Nictitation, or abnormally frequent winking, occurs not infrequently in children as part of a choreic habit.

Stye and Blepharitis.—Stye (hordeolum), small boil on the palpebral margin, and blepharitis, or inflammation of the margin of the lids, while due to infection, may be predisposed to by an error of refraction or some defect in the general health.

Vaccinal Eruption.—This may appear on the eyelids, occurring at the commissures as a small ulcer with an indurated border and yellow floor.

Chancre.—This may appear as a primary sore, and is generally situated on the palpebral conjunctiva.

Malignant Pustule.—Malignant pustule, or specific anthrax, is seen at times, though rarely, on the lids of those who are exposed to infection from diseased animals or decayed animal matter.

Xanthelasma.—This consists in the formation of small, irregular, opaque, yellowish patches, slightly elevated above the surrounding skin. These areas may either remain localized or the disease may involve the palms of the hands, the flexures of the fingers, and the inside of the mouth.

Chalazion.—Small, hard, painless tumors of the lids are due to obstruction of a Meibomian gland with retention of inspissated secretion.

The Orbit.—Exophthalmos.—Exophthalmos, or proptosis, abnormal prominence or protrusion of the eyeball, is usually occasioned by some disease of the orbit or of the neighboring sinuses which encroaches upon the cavity of the orbit. It is one of the diagnostic features of exophthalmic goitre (see Exophthalmic Goitre), and may also be caused by paralysis of the ocular muscles. It has been seen, though rarely, after spontaneous hemorrhages into the orbit in cases of hemophilia and scurvy. It has been frequently observed in nephritis.

Enophthalmos.—Enophthalmos, or recession of the eyeball, may be the result of exhausting diseases, such as peritonitis, or secondary to some orbital lesion. It is very pronounced in the sudden atrophy of the orbital tissues that occur in cholera from loss of body fluids.

Extra-ocular Muscles.—When all of the muscles are in a state of equal tension, and the visual axes are directed straight forward in the horizontal plane, the eyes are then said to be in the *primary position*. Any deviation from this is known as a *secondary position*, the simplest of these being direct lateral or vertical movements.

Manner of Detecting Palsies of the Extra-ocular Muscles.—Normally the eyeballs move in perfect unison and harmony, so that the images of objects fall upon corresponding points of the retina, and single vision obtains. If this harmonious action be interrupted by paralysis of one or

more of the extra-ocular muscles, however, limitation in the movement and deviation of the affected eye result, coupled with double vision, or diplopia.

Limitations in the Movements and Deviation of the Affected Eye.—In studying limitations of motion in the eyes, the examiner seats himself before the patient and requests the latter to follow with his eyes the movements of a candle which is carried through all the different meridians of the visual field, any muscular deviation being made evident by a failure in correspondence of the images from the candle reflected from the cornea, as well as by the lagging in the movements of the eye, owing to the deviation of the action of the affected muscle. Three general laws have been formulated which should be borne in mind in this connection: (1) The limitation in motion as well as the diplopia increases toward the side of the affected muscle. (2) The secondary deviation (the deviation which the sound eye makes while the affected eye is fixing the candle) is greater than the primary deviation (the deviation of the affected eye while the sound eye fixes). (3) The image formed on the retina of the affected eye is projected in the direction of the paralyzed muscle.

Diplopia.—The character of the diplopia varies according to the muscle or muscles whose function has been disturbed. Generally speaking, diplopia is either simple or homonymous, or crossed or heteronymous. In the former the image seen by the affected eye lies on the corresponding side and betokens convergence of the visual axes, while in the latter the image seen by the affected eye is projected to the opposite side and indicates divergence of the visual axes. In order to ascertain the relation of the two images to the respective eyes, it is essential that the diplopia should be carefully tested.

Test for Diplopia.—For this purpose the patient is seated in a darkened room with a red glass placed before one of the eyes in order to facilitate the identification of each image by its color, and a lighted candle is held on a level with the head about five meters off. Having noted any deviation which the eyes make in the primary position the examiner moves the candle through the different meridians of the visual field, the patient being requested to regard the flame with both eyes while the head remains stationary, each deviation being carefully noted.

Homonymous Diplopia.—This is caused by paralysis of the right externus if the images separate on a horizontal plane as the candle is moved to the right; by paralysis of the left externus if the separation occurs as the candle is moved to the left.

Heteronymous Diplopia.—This is the result of paralysis of the right internus if the two images separate in the horizontal plane as the eye looks to the left; the left internus as the eye looks to the right.

Vertical Diplopia.—If one image is higher than the other, it is the result of paralysis of the superior rectus or inferior oblique when the higher image makes an angle with the lower. Paralysis of the inferior

rectus and superior oblique results in the lower image being at an angle to the upper one. In paralysis of the two recti the false image lies to the inner side of the true image; in paralysis of the obliques to the outer side of the true image.

Additional Symptoms.—In addition to the study of the anomalies in motion and of the diplopia, considerable information may often be gained by noting the position of the head in ocular paralysis. Thus, in paralysis of the sixth nerve the face is turned toward the paralyzed side; in paralysis of the fourth nerve it is turned downward and toward the shoulder of the paralyzed side; and in paralysis of the third nerve the face looks toward the shoulder of the same side. Not rarely dizziness is complained of, and there is false projection of the field of vision, causing faulty estimation of distance.

The Clinical Significance of Disturbances in the Motility of the Extra-ocular Muscles.—In addition to the relation which paralysis of the muscles bears to lesions of the brain and of the cranial nerves, and which will be dwelt upon later, diplopia may proceed from some much less serious disturbance, as, for example, derangements of the digestive organs or abuse of alcoholic intoxicants, or from overdosage of belladonna, conium, and gelsemium. Transient attacks of diplopia may be among the earliest symptoms of tabes dorsalis, and may occur at the very beginning of cerebral meningitis.

Monocular diplopia is a rare symptom, and when it can be dissociated from some local disturbance in the media of the eye, may be due to hysteria.

Ocular deviation or *paralytic squint*, which has just been described, must be differentiated from *concomitant squint* or *strabismus*. In this latter variety there is no great restriction in movement of the eyes in any one direction, the faulty position of the visual axes remaining constant while the eyes are moved from side to side, and the secondary deviation being equal to the primary. This is the condition which is commonly known as cast or cross-eye, and usually makes its appearance in children with high degree of hypermetropia.

Nystagmus is a spasmodic condition of the muscles of the eye, producing rapid oscillations of the ball, usually horizontal, sometimes rotary, and rarely vertical. It is of great value as a symptom, being present in many brain lesions, usually those of the restiform bodies, the vermiform process, and of the cerebellum. It is also seen in about one-half of the cases of disseminated sclerosis, in Friedreich's ataxia, in miners, and often as the result of visual defects.

Muscular Insufficiencies.—Of late years much attention has been given by ophthalmologists and neurologists to the study of *errors in the extra-ocular muscle balance* in different reflex psychoses. While the assertion that chorea and even epilepsy may be originated by such deviations is extreme, it is nevertheless quite true that many forms of headache, of vertigo, of nausea, and of vague neuralgic pain of a cephalalgic type can be traced to this source. It is important, therefore,

that the clinician should be acquainted with such errors and with the methods employed for their detection.

Test.—The device of Maddox is usually employed for this purpose. It consists of a glass cylinder, which is fitted into a linear opening in a metallic disk. The patient is seated before a candle flame, five meters away from the examiner, and requested to fix the flame with both eyes. The rod is then placed before one of the eyes perpendicularly and an image of a perpendicular streak of light obtained from that eye. If the streak of light be deviated toward the same side as the eye before which it is held, a condition of excessive convergence or *esophoria* is present, but if the streak deviates toward the opposite side then a divergence of the visual axes or *exophoria* exists. If the streak be on a higher or lower level than the flame, vertical imbalance or *hyperphoria* is present. Balance of the muscles is known as *orthophoria*.

The Conjunctivæ.—The conjunctiva being a transparent, vascular membrane, any changes in the quantity or constitution of the blood will at once manifest themselves in its folds. Thus in anemia there is always pallor of the conjunctival vessels, while in plethora there is usually a passive dilatation of the vessels, which gives the eye an injected appearance and occasions the "bloated eye" of the drunkard. In jaundice the conjunctiva is yellow. In anemia and chronic Bright's disease it is often a dead white. Spontaneous hemorrhages into the membranes are seen in whooping cough, asthma, epilepsy, and in arteriosclerosis, and it may be the seat of hemorrhagic infarcts in ulcerative endocarditis or a hemorrhage may occur after a fracture of the anterior fossa of the base of the skull.

Inflammation of the conjunctiva is an early symptom in measles, and in typhus fever it is a constant sign and serves to distinguish this affection from typhoid. It is also present in yellow fever, and may likewise constitute one of the earliest signs of meningeal and cerebral diseases. A passive hyperemia follows disease of the cervical sympathetic.

The Cornea.—The cornea being an avascular membrane which derives its nourishment from the surrounding structures, is very prone to undergo inflammation whenever the vitality of the system becomes much lowered, and as a result of this inflammation *opacities* remain which have a very deleterious effect upon vision. These opacities may be either superficial or interstitial. When superficial they are not infrequently the result of burns, traumatism, and extension of inflammation from the surrounding conjunctiva; in many cases they denote, however, that the eye has been the seat of a phlyctenular conjunctivitis, a form of ocular disease which is quite common in scrofulous children and in individuals below par.

Superficial *ulceration* of the cornea is observed at times in severe fevers of a typhoid type, measles or scarlet fever. It may result from disease of the conjunctiva, lacrymal duct, or nasopharynx.

Abscesses of the cornea form in the stage of desquamation of variola, and must be differentiated from those which arise in the pustular variety of the disease at an earlier period.

The type of *interstitial opacities* of the cornea, a characteristic haze, is seen in inherited syphilis. Malaria and scrofula may also produce a similar form of corneal inflammation. The small areas of opacity which form in the upper and lower parts of the cornea near the limbus, and which at times encircle the cornea, are known as *arcus senilis*, and are without diagnostic significance. A somewhat similar condition is sometimes due to inflammatory conditions.

After lesions of the fifth nerve the cornea may ulcerate from traumatic and trophic causes; and may suffer from exposure due to inability to close the lids, after paralysis of the seventh nerve, or when there is pronounced exophthalmos.

Iris.—Inflammation of the iris is a common symptom of secondary syphilis; it occurs in the form of a gummatous infiltration of the membrane in the tertiary variety, and is seen, though rarely, in inherited syphilis. It is not an infrequent symptom of gout, and may be caused by tuberculosis, arthritis deformans, diabetes, and gonorrhea.

Uveitis.—Inflammation of the whole uveal tract occurs in the same conditions.

The Pupil.—The average size of the pupil ordinarily in diffuse daylight is 4 mm. in diameter. The pupil normally undergoes certain changes under the influence of certain stimuli. It may react either directly or indirectly to *light stimulus*. In order to observe this, the patient is seated before a window and requested to gaze at the sky. The examiner, stationed in front of the patient with his back to the window, excludes one eye by placing his hand over it, and notes the size of the pupil under diffuse daylight. The eye is then covered with the other hand, and the dilatation which should follow is also estimated. The hand is then withdrawn, and, if nothing prevents, the pupil resumes its original size. The fellow eye is then tested in the same manner. This is known as the *direct reflex action* of the pupil, *indirect or consensual reflex action* being the contraction or dilatation which occurs in the shaded eye when the exposed eye is being examined, and should correspond precisely with the movements of the pupil of that eye.

After the reaction of the irides to light stimulus has been noted, the patient is directed to transfer his gaze to the examiner's finger, which should be made to approach the eye slowly, while its fellow is screened off as in the preceding test. The degree of contraction induced by this *accommodative effort* is carefully noted, and the same procedure repeated with the fellow eye. The obstructing hand is finally removed, and the patient being requested to look fixedly at the tip of the examiner's finger with both eyes, observation is made of the contraction of the pupils, which should be induced by the *effort at convergence* occasioned by approximating the finger to the eyes in the median line.

Dilatation of the pupil also occurs upon stimulation of the sensory nerves of the skin of the cheek or neck.

Hippus.—Hippus is a spasmodic alternating contraction and dilatation of the pupil occurring at times after light stimulus, and is seen at times in general paresis, hysteria, epilepsy, meningitis. Rhythmical alterations in the size of the pupils occur frequently during Cheyne-Stokes respiration, the pupil contracting during the period of apnea and dilating with the first few respirations.

Modification in the Size and Behavior of the Pupils as the Result of Disease.—Pupillary reaction to light is a reflex phenomenon, the optic nerve being the afferent nerve, and the third nerve the efferent nerve supplying the sphincter of the iris, communicating fibers between the corpora quadrigemina and the centre for the third nerve making such a reflex possible. The mechanism of pupillary reaction being of an extremely complicated nature, and necessitating the activity of a number of nerves and nuclei, it is not strange that anomalies in its behavior should frequently be met with in disorders of the central nervous system.

Dilatation of the Pupil (Mydriasis), apart from local diseases, of which glaucoma is the type, may occur in certain psychical states, such as fright and emotion; or it may be caused by disease processes giving rise to irritation of the pupil—dilating centre or fibers (irritative or spasmodic mydriasis), or by paralysis of the pupil—contracting centre or fibers (paralytic mydriasis or iridoplegia).

Irritation mydriasis occurs (a) in hyperemia of the cervical portion of the spinal cord and in spinal meningitis; (b) in the early stages of new growths in the cervical portion of the cord; (c) in cases of intracranial tumor and other diseases causing high intracranial pressure; (d) in the spinal irritation of chlorotic or anemic persons, after severe illness, etc.; (e) as a premonitory sign of tabes dorsalis; (f) in cases of intestinal worms, from irritation of the sensory nerves of the bowels, and sometimes in other forms of intestinal irritation; (g) in psychical disturbances—e. g., acute mania, melancholia, progressive paralysis of the insane (in the last-mentioned disease often unilateral, with myosis of the other eye). (After Swanzy.)

Paralytic mydriasis (iridoplegia) may be due either to a paralysis of the pupil-contracting centre or to failure of the stimulus being conducted from the retina to that centre. It may be found (a) sometimes in progressive paralysis in which at first there was myosis; (b) in various disease processes at the base of the brain affecting the centre for the third nerve; (c) in a late stage of thrombosis of the cavernous sinus; (d) in orbital disease associated with pressure on the ciliary nerves. (After Swanzy.) It is said to be present in acute dementia, when there is edema of the cortex, and may be found in cerebral softening. It results from irritation of the cervical sympathetic and occasionally occurs in aortic insufficiency.

Contraction of the Pupil (Myosis).—After myosis from local causes, especially from the sequels of iritis, has been excluded, it will be found that contraction of the pupil may be caused by a disease process irritating the pupil-contracting centre or nerve fibers, or by one causing paralysis of the cervical sympathetic or by a combination of both.

Irritation myosis is found in (a) the early stages at least of all inflammatory affections of the brain and its meninges, hence in simple, tuberculous and cerebrospinal meningitis (when, in these diseases, the medium myosis gives place to mydriasis, the change is a serious prognostic sign, indicating the stage of depression with paralysis of the third nerve); (b) in cerebral apoplexy the pupil is at first contracted, according to Berthold, who points out that this contraction is a diagnostic sign between rupture of a bloodvessel and embolism, as in the latter the pupil remains unaltered; (c) in the early stages of intracranial tumors situated at the origin of the third nerve or in its course; (d) at the beginning of an hysterical or epileptic attack; (e) in tobacco amblyopia, probably from stimulation of the pupil-contracting centre by nicotine; (f) in persons following certain trades, as the result of long-maintained effort of accommodation (watchmakers, jewelers, etc.), the pupil-contracting centre being subject to an almost constant stimulus; (g) as a reflex action in ciliary neurosis: consequently, in many diseased conditions of those parts of the eye which are supplied by the fifth nerve. (After Swanzy.)

Paralytic myosis occurs in spinal lesions above the dorsal vertebra, such as injuries and inflammations, especially of the chronic form. In the simple form of this myosis the pupil is moderately contracted and reacts to light and accommodation. In tabes the pupil is contracted and responds to accommodation but not to light, a sign of great importance, the so-called *Argyll-Robertson pupil*. Myosis and failure of the light reflex also occurs in general paralysis of the insane and cerebral syphilis.

In acute mania the pupil is usually much dilated; and when this mydriasis changes to myosis, approaching general paralysis may be predicted. Myosis, following on irritation mydriasis, is also found in myelitis of the cervical portion of the cord. If paralytic myosis occurs in bulbar paralysis, the disease is probably complicated with progressive muscular atrophy or with sclerosis of the brain and spinal cord. Myosis may also be due to paralysis of the cervical sympathetic, resulting from injury, from pressure of an aneurysm of the carotid, innominate or aorta, or from pressure of enlarged lymphatic glands. In apoplexy of the pons Varolii myosis is present, but it is not yet certain whether it is an irritation or a paralytic myosis. Certain drugs, as opium and its derivatives, chloral, aconite, and physostigma, also cause myosis.

Inequality of the Pupils. This may denote a lesion of the second or third nerve, an affection of the cervical sympathetic, as in irritation by aneurism of the innominate, general paralysis of the insane, or some unilateral lesion of the brain or upper portion of the cord. Inequality

of the pupil is a frequent finding in apical pulmonary tuberculosis, the inequality apparently having no relation to the side of the lesion. It is also seen in exophthalmic goitre.

The Lens.—Cataract.—An opacity of the crystalline lens occurs as a congenital or senile condition. It may appear in systemic conditions that interfere with the nutrition of the lens, as diabetes and arteriosclerosis or in trauma and other disorders of the eyes.

The Eye-ground.—In order to study the remaining structures of the eye it is necessary to have resource to the *ophthalmoscope*. The ophthalmoscope, in addition to giving information in regard to the condition of the media of the eyes, as, for example, of the existence of commencing cataract, or of opacities within the vitreous humor, unfolds to our gaze the head of the optic nerve as well as the retina and the choroid, and renders patent to our view the different diseases to which they are liable.

Retinitis.—The systemic affection, which is accompanied by a lesion of the retina more often than any other, is disease of the kidneys, especially chronic interstitial nephritis. Indeed, about 30 per cent. of all cases of this variety of renal lesion have an ocular manifestation. Retinitis may also be seen as an early symptom in nephritis of scarlet fever and pregnancy. There is usually associated a mild degree of papillitis with retinitis, a neuroretinitis. Its occurrence in subjects with a cirrhotic kidney is of gloomy import, for patients with a retinal complication in this disease usually die within two years of its first appearance. Retinitis may also be occasioned by pernicious anemia, leukemia, diabetes, syphilis, and heart disease, and by extension of disease, from the ciliary body or choroid or from the optic nerve.

Retinal Bloodvessels.—Hemorrhage into the retina occurs most frequently as a result of rupture of a sclerotic bloodvessel. It may also occur in organic heart disease, purpura, scurvy, grave anemia, diabetes, pyemia, septicemia, malaria, and more especially in the retinitis of nephritis. An embolism may lodge in one of the vessels causing complete blindness if occluding the central artery; partial blindness of a small area if obstructing a smaller vessel. Arteriosclerotic changes in the retinal bloodvessels are usually readily diagnostic and are splendid evidences of sclerotic changes in the general vascular system.

Choroiditis is usually the result of syphilis; but in rare instances the choroid may be the seat of tubercles. Gout may also originate a subacute inflammation of the membrane.

Optic Neuritis.—Inflammation of the optic nerve may affect the head of the nerve (papillitis) or portion of the nerve behind the orbit (retrobulbar neuritis).

Papillitis, or choked disk, is rarely idiopathic, but is occasioned by cerebral growths, by meningitis, especially of the base of the brain, or by anything that causes an increase in intracranial pressure, and by the same constitutional diseases which originate retinitis. It

also occurs in acute fevers, in disease of the orbit, particularly retinitis, in lateral and cavernous sinus thrombosis, and it may be the result of suppression of the menses. Usually, however, choked disk is the result of an intracranial tumor, occurring in 90 per cent. of all such cases, and is an early sign of this disorder.

The variety of optic neuritis just described is an ascending neuritis, the inflammation beginning at the intraocular termination of the nerve and spreading upward to the brain. Retrobulbar neuritis is an interstitial or descending neuritis which is frequently seen in meningitis and which is commonly caused by overuse of alcohol or tobacco, although it may be originated by quinine, the salicylates, lead, or iodoform. It may also be caused by rheumatism, gout, exposure to cold, orbital disease, malaria, and there is a rare form in which the disease is transmitted through certain families from generation to generation.

Optic Atrophy.—This may be secondary to some inflammation of the optic nerve or retina, or it may be a primary disease.

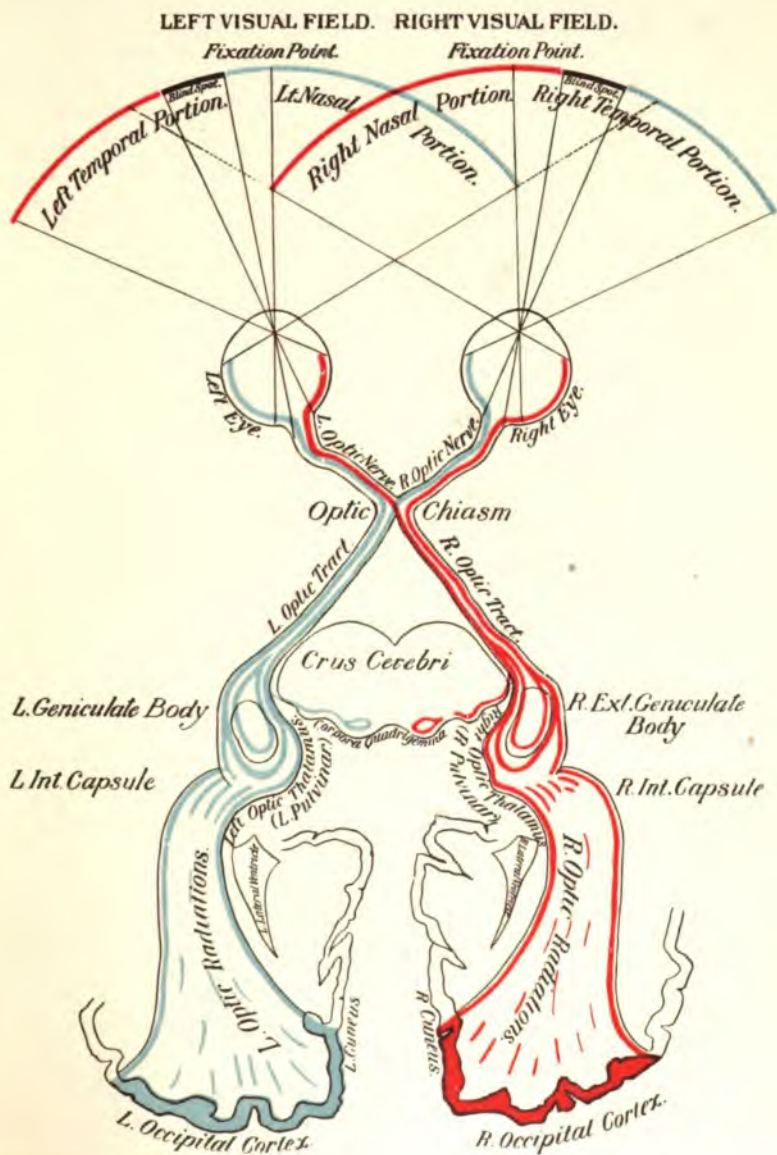
Secondary or consecutive atrophy is usually the result of optic neuritis; it may, however, be originated by local causes either within the eye or the orbit. *Primary atrophy*, on the other hand, though occasionally idiopathic, is generally found associated with some disease of the spinal cord, especially with tabes dorsalis. In this affection it is frequently an early sign, and it has been noted by Benedikt, of Vienna, that when it occurs the patient rarely becomes ataxic. It has also been remarked that cases in which blindness is well advanced, suffer but little from the characteristic pains of tabes dorsalis. Simple atrophy occurs also in lateral and insular sclerosis, and is frequently seen in general paralysis of the insane.

Vision.—Imperfect vision may be due to errors of refraction, to opacities of the transparent media of the eye, to disease of the retina, optic nerve, or central nervous system, or to a neurosis.

Central vision is tested by means of black letters printed on a white test-card, those devised by Snellen being usually employed on account of the admirable system upon which they are founded. The patient is seated five meters away from the card, and, one eye being blindfolded, he is requested to read the lowest line of letters that he can distinguish. If the vision fails to correspond to the standard, it is necessary to exclude hypermetropia, myopia, and astigmatism by means of convex, concave, and cylindrical lenses before it can be definitely asserted that the vision is lowered as the result of disease.

Peripheral Vision, or the extent of space of which the eye is conscious when it is fixed on any given point, may be estimated in several ways; it is accomplished, however, most accurately by means of the perimeter. This instrument consists of an upright rest for the chin and a semi-circular arc or bar, graded in degrees, which revolves on a central pivot, and is capable of describing a hemisphere in space. The eye under examination being directed straight ahead at the fixation-point,

PLATE I



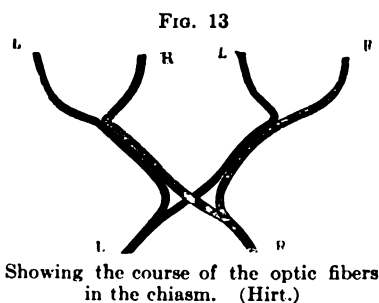


the fellow eye being blindfolded, the test object (consisting of a small square of white paper) is brought from the periphery toward fixation. The patient is then asked to indicate the instant the object is perceived, and the examiner marks the degree upon a chart provided for the purpose. The field for form or white extends over 150 degrees horizontally and 110 degrees vertically, that for the different colors falling within this in the following order—yellow, blue, red, and green.

Scotoma.—As the patient's macula corresponds to the fixation-point in the visual field, the physiological blind spot which is occasioned by the entrance of the optic nerve into the eye will be found in the temporal portion of the field. Pathological blind spots are known as scotomas, and may be either central, paracentral, or disseminated. When central they indicate disease either of the macula or of the fibers of the optic nerves supplying the macula, so that a central scotoma is one of the diagnostic features of retrobulbar neuritis.

Hemianopsia.—This term is used to designate a defect in one-half the field of vision, the defect being named according to the blind area. Thus temporal hemianopsia means that the eye cannot perceive objects situated in the outer half of the field. The most common form of hemianopsia is loss of the temporal field in one eye and of the nasal field in the other, this condition being known as homonymous hemianopsia. If the temporal portions of both fields are lost, the defect is known as bitemporal hemianopsia; binasal hemianopsia indicating loss of the nasal fields of both eyes. Superior and inferior hemianopsia are very rare. *It is often possible by studying the changes in the visual fields to locate quite definitely the seat of the cerebral lesion.* By a reference to the diagram (Fig. 13) it will be at once evident that a *lesion of the chiasm* would necessarily comprise fibers of the optic nerve, and would occasion bitemporal hemianopsia. Such a lesion may be due to basilar meningitis, periostitis, fracture of the body of the sphenoid, distention of the infundibulum and of the third ventricle, tumors, especially those of the pituitary body, and finally gumma. If the lesion is a gumma, there may be transient recurrent attacks of hemianopsia. The lesion causing superior and inferior hemianopsia is usually in the chiasm also, affecting its superior or inferior portion; these defects in the fields may, however, be caused by symmetrical cortical lesions and by optic neuritis. (See Plate I.) If the lesion affects the outer angle of the chiasm, then monocular nasal hemianopsia is the result.

Lesions of the Optic Tract and Centres.—As shown in Plate I, the optic tract after crossing the crus to the hinder part of the optic thalamus divides into two branches. One branch goes to the thalamus and the



external geniculate bodies, and to the anterior quadrigeminal bodies from which fibers pass into the hinder part of the internal capsule, and, entering the occipital lobe, form the fibers of the optic radiations terminating in the cuneus, the perceptive visual centre, while the fibers of the other branch pass to the internal geniculate bodies and the posterior quadrigeminal bodies.

A lesion affecting the optic fibers anywhere posterior to the optic chiasm will produce homonymous hemianopsia, so that this symptom of itself is of little value in localization. There are, however, certain accessory symptoms which, when taken in conjunction with it, will serve to establish the seat of the lesion in most instances. Thus, in hemianopsia from lesions of the optic tract there is an absence of the symptoms which occur when the cortex is affected—as mind-blindness, word-blindness, etc.; while other symptoms indicating a basal lesion are apt to be present, as for example, implication of the cranial nerves, especially those supplying the ocular muscles. Lesions of the optic tract are also frequently associated with disease of the crus cerebri, so that hemianesthesia or hemiplegia of the opposite side of the body would be associated with the hemianopsia. There is, however, a sign which enables us at once to say definitely whether the lesion be in the optic tract or not, and this is known as the *Wernicke or pupillary inaction sign*. This is elicited as follows: The patient is seated in a darkened room with one eye blindfolded, and is directed to look straight ahead into the darkness. The eye being slightly illuminated by an assistant by means of diffuse light from a plane mirror, which is reflected into the eye from a light placed behind the patient's head, the examiner slowly throws a small beam of concentrated light from a concave mirror upon the blind half of the retina. If the pupil fails to react, the lesion is in the geniculate bodies or in the tract, inasmuch as the failure in the pupillary activity indicates that the lesion must have involved the sensory motor arc of the pupil as well as the visual fibers. Although if present the Wernicke sign is of great value, recent observations have shown that its absence is not conclusive and, furthermore, it is an extremely difficult sign to elicit. Lesions of the optic tract may be due to neoplasms or to tuberculous or gummatous meningitis, or more rarely they may be the result of cerebral softening and hemorrhage. As yet clinical evidence is too meager to make a diagnosis of lesions of the primary optic ganglia—pulvinar, anterior corpora quadrigemina, and external geniculate bodies—possible, although in lesions of the pulvinar two typical symptoms occur, viz., hemianopsia and athetosis, and hemianesthesia may sometimes be present. In like manner, while it is generally believed that lesions of the optic radiations cause homonymous hemianopsia, it has not been definitely proved that these fibers have solely to do with vision.

The hemianopsia is usually assumed to depend upon cortical lesions in the occipital lobe when it is unaccompanied by any of the accessory symptoms which have just been detailed. The chief diagnostic

symptom of a central lesion, however, is what is designated as negative vision, "vision nulle," for in these cases the patient has no subjective sensations of the defect in his visual field. Cortical hemianopsia may also be incomplete, one quadrant only of the field being lost and mind or word-blindness may also be present.

Transitory hemianopsia, or *scintillating scotoma*, is the occurrence of symmetrical defects in the field of vision which usually conform to the hemianopic type, and in which a play of lights frequently appears as a precursor of an attack of migraine. (See Migraine.)

Visual hallucinations may also be hemianopic in character, and are due to irritation of the visual memory centre.

Hysterical amblyopia may manifest itself either in complete blindness or in central scotoma, but more commonly as defective central vision with concentric contraction and reversal of the visual fields.

Paralysis of the Motor Nerves of the Eyeball.—Paralysis of the orbital muscles may be due to an orbital lesion or to one at the base of the brain; it may indicate a pontine lesion, or it may be originated by causes operating higher up in the cerebrum above the nuclei. In making the differential diagnosis between central and peripheral palsies, it must be remembered that those of central origin are frequently associated with other symptoms denoting intracranial involvement, while peripheral palsies are generally isolated and often complete.

Peripheral Paralyses.—Such paralyses of the orbital muscles are generally the result of either rheumatism or syphilis. When due to the latter disease, they are usually tertiary manifestations, and especially is this likely to be the case if the third nerve is involved, which seems to be singularly prone to be attacked by gumma at the base of the brain. Paralysis of the sixth nerve is frequently of rheumatic origin.

Syphilis is responsible for fully one-half the cases of central paralysis, the gumma affecting either the nuclei of the nerves or the neighboring brain structure, the third or fourth ventricles, or the aqueduct of Sylvius.

Diphtheria usually causes paralysis of the ciliary muscles; it may, however, affect the nerves supplying one or more of the external muscles. Diabetes is complicated at times by paralysis of the external rectus. Influenza, herpes zoster, and whooping cough are also rare causes of ocular palsies. Paralysis of the eye muscles is seen in paretic dementia, bulbar paralysis, and in multiple and posterior sclerosis. In locomotor ataxia the paralysis may be transient and appear at an early stage of the disease. Ocular palsies have also been observed after poisoning by lead, nicotine, sulphuric acid, carbon dioxide, and tainted meat.

Paralysis of the Third Nerve.—In complete paralysis of the third nerve the upper lip droops, the pupil is partially dilated and immovable, and the power of accommodation is lost. The globe is slightly protruded and strongly diverged externally by the two unaffected muscles (the external rectus and the superior oblique).

In incomplete paralysis of the third nerve, as well as in paralysis of the fourth and sixth nerves, the diagnosis is made by a study of the deviations and by the character of the diplopia, which has already been referred to.

There is a peculiar form of intermitting *paralysis of the third nerve*, known as ophthalmoplegic migraine, which occurs in the young and is associated with headache and at times vomiting.

Paralysis of the ciliary muscle, or cycloplegia, follows a lesion of the trunk of the oculomotor nerve or of the anterior part of its nucleus and the power of accommodation is lost. It is quite common as a sequel of diphtheria, and occurs, though rarely, in connection with spinal disease.

Ophthalmoplegia externa and interna refer to paralysis of all or nearly all of the external and internal muscles. As the lesion in this affection is central, it is frequently known also as nuclear paralysis. The *acute form* is due either to an acute inflammatory process in the nuclei or to hemorrhage; while the *chronic* variety depends upon a degeneration atrophy of the nerve nuclei similar to that seen in progressive muscular atrophy and in chronic bulbar paralysis, with which these conditions may be associated. *In conjugate lateral deviations* of the eyes, although the visual axes are deviated from the middle line, yet they remain parallel. The cause is generally a cortical lesion the result of apoplexy. A spasm deviation of the eyes in the same direction occurs as a result of irritative lesions involving the association centres or tracts, and also in hysteria.

The Localizing Value of Paralysis of the Orbital Muscles.¹—Paralysis of the Third Nerve.—Ptosis on the side of the lesion, without paralysis of the other branches of the third nerve, has been seen in disease of the pons, and again, by forming a factor in crossed paralysis, may seem to localize a lesion in the crus cerebri, although when the third nerve is paralyzed by a lesion in this situation it is usually involved as a whole.

Crossed hemiplegia is a term used to express disease of the crus cerebri when there is paralysis of the third nerve on the side of the lesion, with hemiplegia, hemianesthesia, and often facial and sometimes hypoglossal paralysis of the opposite side of the body. Complete paralysis of every branch of the third nerve without any other paralysis is almost always basal; so, also, are those cases in which hemiplegia, when present, is slight as compared with the degree of the third nerve paralysis. Lesion of the interpeduncular space and thrombosis of the cavernous sinus also produce third nerve palsies; but in the latter the other orbital nerves, as well as the fifth and the optic nerve, may be involved as well. Third nerve paralysis may also be a distant symptom of tumors of the cerebral hemispheres, more particularly if the palsy is accompanied by violent general head symptoms.

¹ This section is an epitome of the excellent article on the subject in Swanzy's *Hand-book of Diseases of the Eye*.

Paralysis of the Fourth Nerve.—As a symptom of cerebral lesion *solitary paralysis of the fourth nerve* is rare. It is generally due to a basal lesion. In combination with paralysis of the third nerve it speaks for a lesion in the cerebral peduncle extending back to the valve or Vieussens.

Paralysis of the Sixth Nerve.—When *paralysis of the sixth nerve* occurs as the only focal sign it is probably due to disease of the base. On account of the lengthened course these nerves take over the most prominent part of the pons, which renders them readily affected by distant pressure, they more frequently present a distant symptom than other cranial nerve. Thus paralysis of this nerve is not infrequently a distant symptom of tumor of the cerebellum, whereas paralysis of the third nerve is more likely to be a distant symptom of a cerebral lesion.

Paralysis of the sixth nerve, when simultaneous in its onset with hemiplegia of the opposite side of the body, indicates a lesion in the pons, usually a hemorrhage, on the side corresponding to the paralyzed nerve. Basal paralysis of the nerve is frequently double, especially in syphilis. In combination with paralysis of the facial, paralysis of the sixth nerve is referable to a pontine lesion.

THE EAR

Subjective Symptoms.—Buzzing, roaring, hissing, singing, and other sounds in the ear—*tinnitus aurium*—are symptoms that may or may not be due to disease of the ear. When they are associated with vertigo, *Ménière's disease* should be thought of. They may constitute the *aura* preceding an epileptic attack or the subjective phenomena attending syncope. Many drugs (*e. g.*, quinine, salicylates) when pushed to the physiological limit cause tinnitus. Anemia, toxemic conditions, and neurasthenic states are frequent causes of ear sounds.

The External Ear.—The thin ear may show the anemia or chlorotic hue more strikingly than any other portions of the body, or cyanosis may be more vividly shown. A dark blue or black discoloration of the cartilage is frequently seen associated with alkaptonuria (ochronosis).

Hematoma auris is seen in general paralysis of the insane and in other forms of insanity. It is a trophoneurosis. The ear is thickened and deformed from the effusion of blood between the cartilages and the perichondrium; discolored; and simulates a subcutaneous effusion due to injury. *Tophi* are observed in the external ears of patients with true gout. They are small, gritty accretions seen in the external ear along the margin or in the depressions. They consist of monosodium urate.

Discharge.—The presence or absence of discharge from the ears must be ascertained when cerebral symptoms or symptoms of infection

are present. Middle ear disease is often secondary to scarlet fever, measles, diphtheria, influenza, typhoid fever, and tonsillitis or pharyngitis. It very frequently results in inflammation of the mastoid, which may extend to the sinuses and adjacent membranes of the brain; or the ear suppuration may be the primary focus from which general infection takes place. It may not be possible in all cases to observe the discharge, which may have diminished or disappeared. Tenderness and edema over the mastoid, perforation or bulging of the ear-drum, as well as other inflammatory signs, point to the occurrence of suppuration of the middle ear and mastoid cells. It must not be forgotten that a discharge of blood or cerebral fluid from the ear may take place in cases of fractured skull. The ears must also be examined in cases of coma from injury, or if the origin of coma is obscure.

The Auditory Nerve.—The Hearing.—The hearing must be tested with the voice, a watch, or a tuning-fork. The ticking of a watch should be heard at a distance of about three feet. In some cases the voice may easily be heard, while the ticking of a watch can be distinguished only with great difficulty. The tuning-fork is used to determine by bone conduction whether deafness is due to obstruction or to disease of the auditory nerve. If it is due to obstruction, the vibrating tuning-fork is heard better when in contact with the mastoid process than when held immediately in front of the external meatus (Rinne's test). Obstructive deafness is always due to disease of (1) the external meatus; (2) the tympanic membrane and middle ear, or (3) the Eustachian tube.

Deafness from internal ear disease may be due to affections of the labyrinth—as inflammation, caries, and necrosis—or of the auditory nerve. The tuning-fork is not heard on contact with the skull nor when placed in front of the external meatus. The auditory nerve may be diseased in its course, or the auditory centre may be affected.

It must not be forgotten that certain drugs, as quinine and the salicylates, may cause deafness. Deafness may be an early and premonitory symptom of typhoid fever or cerebrospinal meningitis, and may occur early or late in the course of mumps. Deafness due to *occupation* is worthy of mention. It is not uncommon in blacksmiths, boilermakers, locomotive engineers, and firemen. In some instances the patients can hear better in the noise incident to their work than when the surroundings are quiet.

Hyperesthesia of the Auditory Nerve.—Abnormal acuteness of hearing (oxyecoia) occurs very rarely in certain cases of facial paralysis, and not seldom in hysteria. In some individuals suffering from hemicrania or tic douloureux, and in meningitis, the hearing of certain sounds—for example, high musical notes and whistling—is accompanied by pain. Nervous patients often complain of subjective noises, buzzing, roaring, hissing, and singing—the so-called tinnitus aurium.

Paralysis of the Auditory Nerve.—No case of absolute unilateral deafness, due to a focal lesion in a hemisphere, has as yet been observed.

Deafness from disease of the auditory nucleus is very rare. That due to disease of the peripheral nerve is much more common. We may have an auditory paralysis similar to that of the facial nerve, or the deafness may be due to pressure from a tumor or inflammatory exudate at the base of the brain, or disease of the mastoid process of the temporal bone. The localization of the lesion is often extremely difficult. The only positive point is, that labyrinthine disease is apt to be accompanied by vertigo, while in disease of the nerve trunk vertigo is absent.

Ménière's Disease.—*Aural Vertigo.*—We may define aural vertigo as a subjective feeling of motion referred by the patient either to his own body or to surrounding objects, with loss of equilibrium and without unconsciousness. The symptoms are paroxysmal vertigo (sometimes so sudden and intense as to throw the patient to the ground), tinnitus aurium, nausea, pallor, clammy sweat, and vomiting. The severity of the attacks varies greatly. There may be momentary unconsciousness; sometimes jerking of the eyeball, nystagmus, or diplopia is present. The disease is paroxysmal in character, but a slight degree of vertigo and tinnitus often persist between the attacks. Some deafness is present. The attack may vary in frequency from several in a day to only one in several months.

Hysterical or Functional Deafness.—This is recognized by (1) its association with undoubted symptoms of hysteria; (2) its sudden occurrence after shock, emotional disturbance, or trauma; (3) the absence of a cause in the auditory apparatus for the deafness; (4) impairment of bone conduction and aërial conduction to the same degree; (5) the frequent coexistence of anesthesia of the pinna and external meatus; (6) the tendency to sudden recovery.

Hysterical Deaf-mutism.—This is a rare condition, characterized by (1) sudden origin; (2) absolute aphasia and aphonia; (3) absence of signs of paralysis of the lips and tongue and of any paralytic phenomena except hysterical hemiplegia; (4) preservation of intellectual faculties and power of writing; (5) frequent coexistence of hysterical stigmas; (6) usually rapid recovery.

CHAPTER XII

EXAMINATION OF THE EXTREMITIES

THE NAILS

Shape.—The appearance of the nails enables us to estimate the duration of certain diseases, or the time when convalescence began; it also indicates local interference with the nutrition of the parts. Thus *curving* of the nails, with clubbed fingers, occurs only in chronic diseases, as phthisis or emphysema or in chronic cardiac disease and aneurism. In the latter it is sometimes found on one hand only. The nails may curve transversely or longitudinally. This latter incurvated change in shape may occur without clubbing of the fingers. The shape is altered in acromegaly and pulmonary osteo-arthritis.

FIG. 14



Clubbed fingers with curved nails, middle finger slightly flexed. (Original.)

Color.—*White marks* or *transverse grooves* on the surfaces are usually seen after trauma, after an illness, and may indicate the date of recovery as they develop at the root of the nail during the illness and take about six months to reach the end of the nail.

Pallor.—We get a good idea of the condition of the blood in the capillaries from the appearance of the tissue under the nails. If there

is anemia, pressure on the finger tips will drive the blood from the capillaries. The *purplish* and *bluish-black* discoloration of *cyanosis* previously referred to is first seen under the nails.

Capillary Pulsation.—Sometimes the *capillaries pulsate*, and this pulsation is readily visible under the nails if slight pressure is made upon their end. It occurs most frequently in aortic regurgitation, after hemorrhages, and in anemia, but may appear in other disorders less frequently.

Inflammatory Changes.—The nails undergo chronic inflammation with destruction in various skin affections, and the matrix is the seat of acute inflammation in onychia, which may be simple or syphilitic, with ulceration, or with loss of the nail and necrosis of the bone.

Trophic Changes.—The nail becomes dark and brittle in *neuritis*, curved in its long axis, and the growth arrested, while lateral arching takes place. The cutis underneath thickens and the skin at the base retracts. The fingers may be clubbed. When growth is resumed a distinct roughened line of demarcation is seen. In leprous neuritis there is destruction of the nails and of the distal phalanges. *Atrophy* and *ulceration* at the base of the nails, followed by necrosis of the phalanges, is seen in the so-called Morvan's disease, which is not really a disease, but a symptom of neuritis or syringomyelia. *Enlargement* with thickening and sometimes twisting occurs after fevers, as typhoid; in the course of syphilis; and in sclerodactyly. The nails in some cases of Raynaud's disease become dry, scaly, and cracked, or hypertrophied. The *growth* is arrested on the paralyzed side in the hemiplegia from cerebral apoplexy. This is tested by staining the nails of the two hands at the same level with nitric acid. Return of functional power is indicated by renewed growth. *Deformity* of the nails (toe) occurs in acute and chronic myelitis. In *locomotor ataxia* the nails sometimes fall out.

THE FINGERS

In gout and arthritis deformans the joints of the fingers are *enlarged* and *painful*. The swelling of the joints of each condition cannot well be distinguished. In gout, *tophi*, hard, white, sometimes glistening masses are likely to be present in the joints or along the tendons, on account of great accumulation of monosodium urate. They are more prominent on the dorsal surface of the joints, and sometimes break through the skin, so that the chalk-like concretion exudes.

Heberden's Nodes.—The term "end-joint arthritis" is also applied to this condition. The nodules develop gradually and are exostoses of the articular tubercles of the distal phalanges occurring as a type of the hypertrophic form of arthritis deformans. At first the joints may be a little swollen and tender. The swelling and tenderness may be periodic and the size may be increased with each fresh paroxysm.

AL DIAGNOSIS

ace. The nodules are often considered re said to be a sign of longevity. It is certain that the large joints are rarely involved when these nodes are present.

Clubbed Fingers.—The tips of the fingers may be *bulbous*, or *club-shaped*, in some cases of phthisis and other forms of chronic lung disease, and also occasionally in chronic heart disease particularly when congenital. The clubbing is associated with changes in the nails.

Deviation in Position and Shape.—*Eversion* is somewhat characteristic of arthritis deformans; but deviations due to abnormal flexion or extension are the most characteristic digital features of the disease.

Flexion of the first phalanx of the little finger is due to contraction of the palmar fascia or to paralysis of the common extensor from disease of the musculospiral nerve. *Contraction* of the fascia of the hand, causing more or less flexion of the little and ring fingers, is frequently seen and may be from trauma or an indication of gout, *Dupuytren's contraction*.

FIG. 16



arthritis deformans. (Original.)

Abnormal *extension* is often very marked. *Hyperextension* of the middle phalanx is due to paralysis of the flexor sublimis from disease of the median nerve; hyperextension of the distal phalanges to paralysis of the flexor profundus muscle from disease of the median and ulnar

FIG. 17



Pseudomuscular atrophy. Claw-hand. (Gray.)

nerves. Extension of the proximal phalanx, with extreme flexion of the two distal phalanges, contributes to form the "claw-hand." *Contractions* due to chorea or to central lesions, as posthemiplegic contractions, will be considered under Special Diagnosis.

THE HANDS

Shape.—We bear in mind the variation in *shape* of the hand in different types of individuals—the broad and heavy hand of the sanguine, the slender, dexterous hand of an individual of the nervous temperament, the large joints of the hand of so-called strumous persons, and the effeminate hand of one who is inclined to tuberculosis, present sharp contrasts. Then, too, the occupation hand indicates in a general way the disease the patient is liable to—none more striking than the hand of a miner, the blue-black dottings of which clearly indicates the possibility of anthracosis. Finally, we note the broad hand and clubbed fingers that are seen in congenital heart disease and the thick broad hand of acromegaly. The withered hand of age and wasting of the hands, as in phthisis or malignant disease, need not be referred to, as they are part of the general process.

Presenting more striking changes in shape are the peculiarly deformed hands seen in affections of the muscles and joints. These deformities will be described in the respective sections. (1) We have the claw-hand of progressive muscular atrophy, of inflammation of the ulnar and median nerve, and of chronic poliomyelitis, due to paralysis and atrophy of the interossei and lumbricales muscles, causing dorsal extension of the proximal phalanges with flexion of the others. (2) The "*seal-fin*" hand of chronic gout—spasm of the extensor muscles causing deflection to the ulnar side. (3) The "*ape-hand*," due to

wasting of the thenar and hypothenar muscles, when the thumb assumes a position parallel to the fingers. (4) *Deformity*: The deformed hand of arthritis deformans and of gout are characteristic. In the former the tapering, shining fingers, the bulbous phalangeal joints, the pallid, clammy surface dotted with freckles, the locked joints, the atrophied muscles, combined at times with exquisite tenderness of the involved parts, make a picture never to be forgotten. The peculiar deformity occurring in scleroderma is described in the chapter devoted to the Skin. Then we have the deformity resulting from flexion of the hand on the forearm, the forearm on the arm, as seen in *cerebral palsies* of children and in the *hemiplegias*.

Deformities.—Deformities of the hand from other causes than the ones just mentioned are often observed. Temporary *contractures* occur in tetany, in temporary hemiplegia or monoplegia, and in paralysis of the extensors. *Dropping* of the hand from the radius toward the ulnar occurs in acute poliomyelitis from paralysis of the extensors. Then we have paralysis of the median, ulnar, and other nerves, with their characteristic deformity. (See Nervous Diseases.) So-called *wrist-drop* is seen in *peripheral neuritis* (musculospiral nerve), and may be unilateral or bilateral. The hand hangs from the wrist on account of paralysis of the extensor muscles. Both hands may drop, although it sometimes happens that one is affected from a few days to a few weeks earlier than the others.

The “*spade-like*” hands of *myxedema* and the enlarged bones of the hands in *acromegaly* and *pulmonary osteo-arthritis* are described in other sections.

Movements.—One can infer the limitation of movements of the hands in the affections described above. The stiffened and immobile hand of chronic gout, in which enlarged joints are prominent, contrast with the painfully locked hand of arthritis deformans. Involuntary movements, as tremors and spasms, are also observed. The *tremor* of age, of hysteria, of paralysis agitans, of exophthalmic goitre, of mercurial and other intoxications, and of disseminated sclerosis, is most marked in the hands. It is in the hands and arms we see that most significant tremor or twitching with aimless picking at the bed-clothes, known as *subsultus tendinum* and seen in the so-called typhoid state. *Twitching* and *spasm* of the hand or arm are seen in convulsive disorders and may be unilateral or bilateral, as in hysteria, chorea, epilepsy (true and Jacksonian), tetanus, and tetany. When permanent, the convulsions are an expression of a chronic cerebral process, as hydrocephalus. Alternating spasm and relaxation of the fingers, hand, and arm are seen in athetosis.

Local Enlargement.—The *swellings* of the hand, inflammatory or edematous, do not differ in cause or appearance from swellings of the joints or of the subcutaneous connective tissues in other portions of the body. Several exceptions are to be noted: (1) The swelling that attends *acute articular rheumatism* with involvement of the wrist-joints

extends over the dorsum of the hand frequently, while the fingers escape; (2) a localized swelling on the dorsum of the hand is often due to a *ganglion* or enlarged bursa of a tendon. (3) Gubler's *tumor* is a swelling that is seen in wrist-drop from displacement backward of the carpal bones; (4) long-contained inflammatory swelling, with subsequent rupture of the skin, is seen in *mycetoma*. Finally, traumatic injuries produce tenosynovitis, bone affections, and palmar abscess. It is important to remember that syphilis and gonorrhea may be causal factors in the production of such processes.

The Skin of the Hand.—The skin of the hand is smooth or rough, dry and harsh, moist and warm, under the same circumstances that affect the skin generally. The dorsal surface and the palm are *moist* and very *soft* and the former is dotted with freckles in arthritis deformans. In progressive muscular atrophy and exophthalmic goitre the skin is *moist*. The cold, *clammy* skin of one laboring under excitement is well known.

Color.—The color of the hands is significant of the state of the circulation and the condition of the blood. The *blue* finger tips and the *pallid* hand accompany similar color changes in the lips, and are early signs of *cyanosis* and *anemia* respectively.

The Circulation.—RAYNAUD'S DISEASE.—LOCAL ASPHYXIA.—The hands or fingers become pale and intensely cold; they are the seat of numbness, and are without sensation. The term "*dead fingers*" graphically describes the appearance. There are alternating paroxysms of a pallid and a livid hue which may occur several times in twenty-four hours. In some cases the lividity becomes so intense that gangrene ensues in small superficial spots, or even involves the whole finger. Pain may or may not be present, and does not increase when the hand hangs down, and is more frequently present and excruciating at the time the fingers are "dead." The tips of the nose and the lobes of the ears may be affected, and occasionally other parts of the surface. The condition is a vasomotor or trophic disorder.

ERYTHROMELALGIA.—Erythromelalgia is a vasomotor or trophic disorder, frequently associated with endarteritis, characterized by redness of the surface with increased temperature, and is usually seen in the extremities. It is worse in summer, increased by artificial heat, and aggravated when the extremity is dependent or pressed upon. The redness is attended by burning and extreme local discomfort, in which all sorts of sensation are described—tearing of the finger nails, pulling or pricking of the skin, twisting of thousands of needles, and other forms of torture.

Glossy Skin is seen after nerve injuries and neuritis, and in central affections in which the trophic nerves are involved. The skin is shiny, smooth, drawn very tightly over the surface, and sometimes atrophied. Red and pale mottling may be seen. The surface is free from hair. Burning pain precedes and accompanies the change. The finger tips become pointed.

THE ARM

The Vessels of the Arm.—Atheroma of the branchial artery and its branches in the forearm is indicated by enlargement, visible pulsation, and tortuosity of the vessels.

Edema of one arm may be due to thrombosis of the axillary vein, as in heart disease, or phlebitis of the smaller veins, or to occlusion of this vein or of the subclavian from external pressure by a mediastinal tumor, aneurism of the aorta, the innominate or the axillary artery; or more commonly enlargement of the axillary glands.

THE FOOT AND LEG

Enlargement or deformities of the feet and legs may be due to changes in the joint, the bones, or the subcutaneous connective tissue. Hence, we would have *swelling* due to edema and myxedema, and enlargements due to acromegaly and pulmonary osteo-arthritis (*q. v.*). *Flat-foot*

FIG. 18



Appearance of the legs in (a case of) leprosy showing pigmentations, dermatitis, ulcers, and amputations.

must always be looked for when inability to walk is complained of. Changes in the shape of the foot from muscular affections, bearing in mind that "claw-foot" is allied to "claw-hand," are found in progressive muscular atrophy and in Friedreich's ataxia.

Three *nutritional changes* take place in the feet that are of diagnostic significance: *perforating ulcer* of the foot, a trophic change occurring in syphilis, neuritis, general paresis, leprosy, locomotor ataxia or diabetes; *gangrene* the result of endarteritis (usually senile) or occurring in the course of diabetes mellitus; *mycetoma*, or "Madura foot." Perforating ulcer usually begins as a blister, and goes through the successive stages to abscess and finally ulceration.

The *nails* of the feet are subject to the same changes that take place in the nails of the fingers.

The Vessels of the Foot and Leg.—Varicose veins follow phlebitis or intra-abdominal pressure from tumors, fecal accumulation, or pregnancy. Phlebitis of the femoral vein is characterized by pain and tenderness of that vessel and by edema of the foot. Deep-seated phlebitis may cause edema and congestion of the foot and knotted veins. The femoral artery is thickened and rigid in arteriosclerosis. Absence of pulsation with localized pain is due to closure from thrombus or embolus, and is followed by gangrene.

The Bones of the Foot and Leg.—Bony growths or nodes are due to syphilis. Local or more general enlargement of the tibia, with pain, occurs in syphilitic periostitis during the tertiary stage and in the hereditary form. Swelling of the periosteum, with redness, heat, pain, and high fever, is due to an infection, and is often followed by suppuration and necrosis. Red, swollen, and indurated areas over the extremities may be due to erythema nodosum, scurvy, leprosy, or some form of purpura.

Cold Hands and Feet.—Coldness of the extremities is a common and often a serious complaint. It is natural to expect a peripheral coldness when there is myocardial insufficiency, also in collapse, in hemorrhage, and in shock. It is a common vasomotor condition in nervousness independent of hysteria. It is a marked feature in true and false angina pectoris. In senile endarteritis coldness of the hands and feet is common. The extremities are often bathed in a cold and clammy perspiration. The poisons of gout, rheumatism, and other diseases may cause cold hands and feet by irritating the peripheral and vasomotor nerves. In gastric and intestinal dyspepsia the absorption of toxins may produce this symptom.

Sensation.—Changes of *sensation* will be considered in the chapter devoted to an examination of the nerves. It is sufficient to state that circumscribed *anesthesia* due to causes limited to the skin is seen in morphea, in the anesthetic form of leprosy, and in certain ischemic states (urticaria). It is accompanied by loss of tactile sensibility. *Hyperesthesia* and *paresthesia* attend various local affections, but save in nervous diseases are without diagnostic significance.

the taking of food, or the onset of fever, tinges the cheek with a hectic flush. In migraine, the burning may be limited to one side of the face. Capillary congestion on the cheeks or on the tip of the nose occurs with the endarteritis of the aged, but is seen also in early life in cases of hepatic cirrhosis or obstruction of the hepatic circulation from other causes. Slight burns and scalds cause a localized redness,

Pallor.—This is caused by diminution of the amount of blood in the periphery or because of changes in the blood.

Temporary Pallor.—Pallor of this type is *acute*, and may or may not be recurrent. It attends fright, syncope, or nausea and vomiting. It occurs also in acute poisoning, in acute disease, such as diphtheria, and in hemorrhage. The pallor due to loss of blood may be instantaneous if the hemorrhage is sudden and large, or develop gradually if it is small and continued over a long period. The onset of sudden pallor is of diagnostic significance in diseases in which hemorrhage may occur, as aneurism, gastric or intestinal ulcer, and typhoid fever. Symptoms of collapse are seen with this form of pallor.

Permanent Pallor.—Pallor of long duration, or *chronic pallor*, if we may so term it, is seen in a number of diseases. It is characterized by gradual onset. The number of red corpuscles is diminished, with loss of the hemoglobin. Pallor is the normal condition in a large number of individuals, particularly in those who lead a sedentary life and are confined within doors. Generally speaking, pallor is characteristic of blood affections; but it is not necessarily present in *leukemia*—indeed, the cheeks and lips may be red. Moreover, *anemic people are not necessarily pale*; and *pale people are not always anemic*. Striking examples of pallor are seen in chronic *Bright's disease*, in *cancer*, in *chronic poisoning*, as from lead or arsenic, in *chronic catarrh* of the stomach or of the bowels, and in *chronic infectious processes*, as tuberculosis and syphilis.

Degrees of Pallor.—*Greenish hue*: while *paleness* is recognized as the fundamental or prevailing color of the skin in many of the above-noted affections, an additional tinge gives a characteristic hue to the skin; thus in *chlorosis* there is a *greenish* appearance of the face, which is in striking contrast to the pearly colored conjunctiva. *Yellowish hue*: in *carcinoma* the *yellowish* tinge often causes the pallor to be mistaken for jaundice. In *pernicious anemia* a *straw-colored* appearance of the skin has been frequently described, which may cause the disease to be mistaken for carcinoma.

The Yellow Skin.—*Jaundice.*—The yellow coloration is seen not only in the skin, but also in the sclera and the mucous membranes. The discoloration of the skin is not difficult of recognition. It varies in shade from a slight yellow hue to yellow green or olive green and in many forms of jaundice to brownish yellow. The yellow hue of the skin in jaundice may be preceded and is always accompanied by tinting of the conjunctiva. The mucous membrane under the tongue early gives evidence of jaundice; or, if the lips are everted and a glass slide

pressed evenly on the surface, the yellow discoloration of the mucous membrane will shine through.

The yellow tint of the conjunctiva must not be confounded with the same color due to subconjunctival fat. The latter is not uniform in the conjunctiva, but may occupy cone-shaped areas.

The Blue Skin.—*Cyanosis.*—This peculiar hue is recognized without difficulty. The bluish or bluish-red appearance of the skin is first seen at points farthest from the central organ of circulation, as in the extremities. It is early seen in the finger tips, particularly underneath the nails, about the phalangeal joints, and in the lips. The mucous membranes also exhibit the change early. Subsequently the entire surface of the skin may become dusky or cyanosed as its cause increases in degree.

When the cyanosis is bilateral or uniformly seen over the surface the cause is general and central, as in the heart or the lungs, or from certain poisons, as the coal-tar preparations, illuminating gas, opium, hydrocyanic acid, and strychnine. When it is unilateral or local a venous trunk is the seat of obstruction.

The causes of general or bilateral cyanosis are (1) respiratory; (2) circulatory; (3) hemic.

1. *Respiratory.*—All conditions which interfere with the aëration of the blood cause more or less cyanosis. Practically, sufficient air cannot get to the blood, or sufficient blood to the air. Obstruction of the air passages, diminution of the respiratory area, and diminished or inefficient respiratory movements prevent oxygen from getting into the blood; interference with the circulation in the lungs prevents the blood from getting air. The two processes are often combined. In short all the factors productive of dyspnea as narrated in the chapter devoted to that symptom are causes of general cyanosis. It is important to remember that the organism may adapt itself to the causes of dyspnea and the symptom abate; such does not seem to be true with the symptom cyanosis. This adaptation may permit of grave cyanosis in tuberculous laryngitis before symptoms of obstruction have set in. Briefly, the respiratory causes are (a) *obstruction of the air passages*: (1) *Faucial* obstruction; (2) obstructive *laryngeal* diseases; (3) *tumors*, pressing on the trachea or bronchi; (4) *spasm* of the bronchi, as in asthma; *occlusion* of the bronchioles, as in bronchitis, particularly the grave forms of bronchopneumonia in childhood, and in *emphysema*; (5) *foreign bodies* in the larynx, trachea, or large bronchi. (b) *Diminution of the respiratory area*, as in *pneumonia*, in *edema* of the lungs, in *tuberculosis*, and in all forms of *pleural effusion* and of *intrathoracic tumors*. (c) *Diminished or insufficient respiratory movements* caused by (1) muscular or pleuritic pain; (2) impaired action of the diaphragm, on account of upward pressure by accumulations in the abdominal cavity, as large peritoneal effusions, an enlarged liver or spleen, or an abdominal tumor; (3) by paralysis in *bulbar paralysis* and *peripheral neuritis*, in *paralysis* of the diaphragm, *spasm* of the muscles of respira-

tion (as in tetanus), and in *progressive muscular atrophy* and in other rare affections of the muscles, as *trichinosis*. (d) *Obstruction of the pulmonary vessels* from pressure on the pulmonary artery or vein by aneurism or mediastinal tumor, or from disease of the heart if secondary pulmonary congestion has taken place from failure of compensation.

2. *Circulatory*.—Interference with the systemic and pulmonary circulation causes a stasis of the blood, with consequent general cyanosis, the result both of insufficient aeration and of damming back of the venous blood in the peripheral capillaries. This is the result of cardiac failure (decompensation), whether due to primary endocardial, myocardial, or pericardial disease. In congenital heart disease there is the added factor of admixture of the arterial and venous blood within the heart. Such cyanosis is often so great that the affection has been termed "blue disease," or *morbus cæruleus*.

3. *Hemic*.—Affections of the blood at times may cause a general cyanosis. These may be due to (1) increase in the cellular elements of the blood, as in polycythemia and chronic illuminating gas poisoning; (2) chemical changes in the blood with the formation of carbon-monoxide hemoglobin or methemaglobin. This latter condition is due to poisoning by such coal-tar derivatives as phenacetin, phenol, and acetanilid, or by potassium chlorate, potassium permanganate, and turpentine. Hydrocyanic acid causes some unknown chemical change in the blood which causes cyanosis.

Local cyanosis is seen when there is obstruction of the venous trunks from external pressure and when there is disease of the venous wall or other conditions causing thrombosis. It may be limited to the head and upper extremities in obstruction of the descending cava by tumor or aneurism or to the lower portion of the trunk and the lower extremities in obstruction of the ascending cava by pressure from tumors within the abdomen or thorax. One extremity may be the seat of local venous stasis from pressure upon the veins, or their occlusion by thrombosis: the arm in cases of cancer of the breast and axillary glands, the leg in cases of femoral phlebitis, represent typical forms of venous stasis. A striking form is due to causes affecting the vasomotor nerves, giving rise to peripheral capillary spasm. (See Raynaud's Disease.)

General Pigmentation.—When the entire surface of the body is bronzed it is usually due to *Addison's disease*, although it must be remembered that extension over the surface may be very gradual, and some regions may be more deeply bronzed than others. It may be due also to that rare condition, hemochromatosis (bronzed diabetes). The pigmented areas in the mucous membrane of the mouth, seen at times in negroes, must not be mistaken for the pigmentation of Addison's disease.

Local Pigmentation.—In *sunburn* the discoloration is uniformly limited to parts that are exposed to the sun, and the mucous membranes are free.

In persons living in filth, general discoloration of the skin takes

place, known as "*vagabonds' disease*," but because it is so general and the skin is rough and thickened, and other evidences of filth are seen, it can easily be recognized.

Chloasma, localized, irregularly shaped, bronzed or yellow-brown discolorations of the skin usually of the face and neck may develop as a result of trauma and inflammatory changes in the skin or may appear in the course of Graves' disease, malaria, syphilis, leprosy, scleroderma, disease of the pancreas or the liver, particularly cirrhosis, chronic gastric ulcer, and in chronic constipation (symptomatic chloasma). The condition is frequently congenital.

In certain cases of *pulmonary peritoneal* and *tuberculosis* a peculiar brown discoloration simulating Addison's disease, but most marked on the chest, is present, the *chloasma phthisicorium*, and is also seen in cases of intra-abdominal neoplastic disease, probably in both cases from disturbances of the chromaffin system.

The melasma that occurs in *uterine disease* and in *pregnancy* (uterine chloasma or melasma gravidarum) resembles somewhat in color the bronzing of Addison's disease, but is usually found only on the face.

Parasitic diseases of the skin, notably *tinea versicolor*, old syphilitic skin lesions, psoriasis when healing and many other skin affections may cause a yellowish-brown or brown discoloration of the skin. Arsenic frequently intensifies the pigmentary changes in skin lesions and may also cause excessive pigmentation elsewhere in the skin if used for a long time. Melanotic pigmentation of the ears, face, or hands may appear in ochronosis, or it may appear anywhere in the skin in melanotic cancer.

Freckles are not usually of special diagnostic significance. Their occurrence in an unusual degree on the back of the hand and forearm has been observed, however, in cases of arthritis deformans.

Argyria.—If silver nitrate is administered over a long period of time, fine black particles of the metal, its insoluble albuminate, are deposited in the kidneys, the intestines, and the skin. The discoloration of the skin is gray or grayish black. Small specks may also be noted in the mucous membrane of the mouth. The cornea and nails are not affected.

Leukoderma (vitiligo) is a condition characterized by the appearance of circumscribed areas, in which there is a deficiency of skin pigment. The spots are irregularly shaped, milky white, and most common on the back.

Hemorrhages into the Skin. Hemorrhages produce discolorations of the skin which vary in hue from deep red to yellow brown, according to their depth beneath the surface, and to the degree of absorption that has taken place since the hemorrhage occurred.

Hemorrhages in the skin are called, according to their size, *petechiae*, *ecchymoses*, *ribices*, and *hematomas*. The petechiae and ecchymoses are apt to appear in the hair follicles, and vary in size from that of a pin-point to that of a split pea. They may be raised above the surface of the skin. They do not disappear upon pressure. Ribices frequently

appear in the form of lines or streaks. Hematomas are tumors containing blood. (See Purpura.)

Eruptions of the Skin.—General Symptoms.—The *subjective symptoms* of any eruption are of great importance in the diagnosis. Pain, itching, burning, smarting, and tenderness are significant of the inflammations. Pain due to inflammation is constant and smarting, burning or throbbing in character. Sometimes, however, pain of a neuralgic character, intermittent and distributed in the course of nerve trunks, precede the development of eruption. This is seen in herpes zoster. *Itching* is an important symptom in diseases of the skin. It is not, as a rule, present in the eruption due to the exanthemas, except in smallpox, chickenpox, and rubella. Its absence is a striking peculiarity of the eruptions of syphilis; but in erythema, especially if associated with edema, it is a most annoying symptom. In other skin diseases, as eczema, psoriasis, and the parasitic affections, it is much more common and extremely annoying.

Itching may be present without any anatomical evidence of skin disease. It is seen in the troublesome *pruritus* that occurs in the aged, particularly about the intestinal and genito-urinary orifices, symptomatic of affections of the organs related thereto. It is a symptom which should lead to an examination of the urine, as diabetes is sometimes found to be the fundamental source of the complaint. It has been previously noted that itching occurs to a high degree in jaundice. It may also be produced by drugs, as opium and morphine, and sometimes quinine.

The following eruptions are of diagnostic value to the internist:

Erythema.—Erythema may be divided into acute, contagious, exudative dermatoses, represented by measles, scarlatina, rubella, and smallpox, and the eruptions of typhoid fever and typhus; and the acute, non-contagious, inflammatory dermatoses, many of which may be confounded with the eruptive fevers. They have been divided into the rubeloids and scarlatinoids. The scarlatiniform erythemas are febrile at the beginning, subacute in course, but of longer duration than the fever they simulate. They are the most common forms, and arise from infectious diseases, such as puerperal fever, septicemia, and gonorrhea; or from toxemia due to drugs or articles of food. Among the important non-contagious varieties of erythema are *erythema multiforme*, *erythema nodosum*, *urticaria*, *herpes*, and *roseola*.

Erythema Multifforme.—In simple form it is seen with papules or with exudation; it may disappear in a few hours, or persist for a day or two and form rings (*erythema fugax* or *erythema annulatum*). With the fading of the redness faint desquamation ensues, and there may be a few pigment marks. The annular form is observed in rheumatic fever. Both varieties may also be found associated with the following infections; typhoid fever, puerperal fever, gonorrhea, cholera, ulcerative endocarditis and osteomyelitis, syphilis, leprosy, vaccinia, and septicemia.



unilateral, and its precise limitation to one-half of the body is of the greatest diagnostic significance.

Severe neuralgic pains localized in the nerves in the distribution of which the eruption takes place may precede the eruption by several days, and persists long after the eruption has subsided. This is particularly the case in old people.

Herpes labialis, or *facialis*, is an eruption of vesicles arranged in groups or clusters upon an inflamed surface. They appear very suddenly upon the upper lip or the alæ of the nose, sometimes on the cheek or chin, and may occupy the inside of the mouth. They undergo the same changes as herpes zoster, but are not attended by severe neuralgic pain. They are also symptomatic of an internal disorder, as an acute coryza, or follow a rigor, as in intermittent fever or pneumonia. They may be present in epidemic cerebrospinal meningitis, but never in tuberculous meningitis. Herpes iris and herpes preputialis have no diagnostic significance of internal disease.

Roseola.—Roseola is an eruption of a deep rose color, not arranged in crescentic patches as in measles, nor scarlet and capable of being resolved into innumerable red points as in scarlatina. It is not so diffuse as the latter. It may precede smallpox, scarlatina, measles, cholera, typhoid fever, diphtheria, and malaria. In smallpox, in cases of cholera, and after parturition and surgical operations the rash is copious, but is characterized by being seated over the lower half of the abdomen and the anterior and inner aspects of the thighs. It may appear elsewhere, but is usually confined to that portion of the body.

Erythema of Special Diseases.—Sufficient reference has been made to the erythema that attends *rheumatic* fever. A few other infectious disorders are associated with the development of an eruption. In *cholera*, during the period of reaction, a rose rash which may resemble erythema, urticaria, or scarlatina appears coincidently with a rise of temperature. It is most frequently seen on the forearms and backs of the hands, but it may cover the back and limbs. It may be slightly hemorrhagic and last two or three days. A slight desquamation usually follows. In *influenza* a roseolous eruption, covering the trunks and limbs and later becoming papular, is seen in rare cases.

Erythematous eruptions are sometimes seen in the course of *Bright's disease*. Two forms are seen: the roseola on the feet, legs, and hands—rarely on the chest and abdomen; and the papular form on the thighs, arms, and shoulders. Itching and other subjective symptoms do not attend the eruption. A form with desquamation may begin on the limbs. These erythemas are common in the later stages of Bright's disease, but are not of ill-omen. In acute Bright's disease a transient roseola is observed very rarely; so also is purpura. If there is much anasarca in nephritis, erythema is more common. The eruptions usually appear independently of uremic symptoms, and disappear during their continuance.

Sudamina.—Sudamina, or miliaria, are small clear vesicles seen in large numbers, usually on the abdomen, but also on any other part that reflects the light strongly. They are seen during and after the subsidence of profuse sweats. While actual perspiration is seen on the forehead, the trunk may appear free from moisture. When the hand is placed over it, as on the abdomen, the dryness is noted, but at the same time a roughened, nutmeg-grater-like sensation is felt. On close inspection this is observed to be due to the eruption just mentioned. The vesicles are usually of good prognostic omen in the course of febrile diseases, particularly typhoid fever.

Medicinal Rashes.—Most of the so-called medicinal rashes belong to the erythemas.

The following drugs are known to cause erythema: potassium bromide and iodide, copaiba, cubebs, the essential oils, capsicum, santonin, chloral, opium, morphine, antipyrin, salicylic acid and its compounds, iodoform, belladonna and atropine, tar, carbolic acid, arsenic, cannabis indica, digitalis, mercury, silver, copper, and diphtheria antitoxin.

Belladonna produces in susceptible persons, or when administered in poisonous doses, a diffuse, bright red erythema, closely resembling that of scarlet fever, but without the interspersed dark red points which characterize the latter. *Atropine* also produces in some persons, especially on the shoulders, arms, chest, and face, an eruption of disseminated, small, hard vesicopapules showing no tendency to pustulation.

The *bromides* produce a characteristic pustular eruption which is most intense upon the shoulders, face, chest, and arms.

The *iodides* produce an eruption which is not often pustular, but an erythematous or papular rash is not uncommon. It appears chiefly about the forearms, face, and neck. Vesicles, bullæ, and purpuric spots are also occasionally seen.

The eruption produced by *quinine* is generally erythematous and is attended with itching and burning. The face and neck are attacked first.

Opium and its alkaloids also produce in susceptible persons an erythematous scarlatinoid eruption which is accompanied by intense itching. Itching, especially about the nose, is much more common without eruption.

Copaiba produces a vesicopapular or papular eruption resembling urticaria and erythema multiforme, which is usually most abundant on the extremities and by itching. The lesions may be purpuric.

The eruption of *cubebs* is a diffuse erythema, with millet-sized papules, coalescent here and there. Unlike the eruption of copaiba, it is more copious over the face and trunk than over the extremities.

Antipyrin causes a measles-like or urticaria-like eruption.

Scars.—Scars are important proofs of the occurrence of previous disease, especially smallpox, chickenpox, and syphilis. Scars of the first

two occur in the form of circular pits and almost always on the face. Scars of syphilis are larger, circular or oval in shape, and seen usually to the best advantage on the extremities, but the single scar on the forehead is strikingly suggestive. Scars upon the legs in persons under thirty years of age, when not traumatic, are almost always syphilitic. Scars as the result of suppurating glands are seen most frequently in the neck, but may be found wherever there are glands, especially under the jaw and in the axilla and groin. The adenitis if in the neck is usually tuberculous; if in the groin, a venereal infection. In both instances, as well as in other glands, infection may have followed erysipelas, measles, or like disorders.

The appearance of the scar indicates its age in a general way, and hence throws light upon the patient's previous history, and also serves as a check upon the accuracy of his statements.

Scars the results of wounds, injuries, or operations, may be seen anywhere; they are of importance only so far as they may furnish a clue to the cause of existing disease. Of such nature are the scars upon the head in cases of brain disease, particularly epilepsy.

The scars of pregnancy, the striae seen upon the lower part of the abdomen and the upper part of the thigh, must not be confounded with similar scars that may remain after a severe attack of edema, and which are sometimes found in fat persons. They are also seen after typhoid fever.

The Nutrition of the Skin.—The color is a fair index of the nutrition of the skin, but further information is obtained by *palpation*. In *health* the skin is smooth, firm, and elastic. When pinched up between the thumb and fingers and then allowed to escape, it slips quickly back into its former position. When pressed or squeezed, it becomes pale from expression of blood, but resumes its natural hue immediately.

As age advances the skin becomes less elastic, and in old persons may lie in wrinkles. When pinched up between the fingers the skin is more inclined to remain wrinkled. Fat persons whose skin is firm and hard are in much better conditions than those whose skin is loose and flabby. The latter condition is frequently met with in babies, particularly those fed on artificial foods. When the skin is thin and dry and has lost its tone, so that when pinched into folds it regains its smoothness slowly and sluggishly, it is usually evidence, in a person under fifty, of some grave cachexia, as carcinoma.

Moisture of the Skin.—*Moisture* and *dryness* are in a sense correlated with the nutrition of the skin. It is quite certain that when the skin is abnormally dry its nutrition is impaired. In health the skin is not perceptibly moist, except as the result of physical exertion or exposure to heat, or as the immediate result of imbibing a hot fluid or a sudorific drug. There is considerable individual difference, however, within physiological limits.

Perspiration Increased.—*Hyperidrosis*.—It may be general or local.

A. GENERAL INCREASED PERSPIRATION.—1. Generalized sweating with fever occurs in the course of rheumatic fever, when the sweats are strong in odor and acid in reaction. It is seen in *tuberculosis*, especially in the miliary variety. It is sometimes marked throughout the course of *typhoid fever*. General perspiration also attends the violent muscular action of *tetanus*, but is not seen in *epilepsy*. An example of general sweating is seen in that curious affection to which the term "*sweating sickness*" has been applied. Sweating is extreme in *trichinosis*.

2. Afebrile sweats occur from excitement or slight exertion during *convalescence*. A general profuse perspiration may be of short duration and occur suddenly after fright or as a result of shock. Sweating frequently accompanies severe pain, and is a common symptom of hyperthyroidism.

More striking still is the perspiration that suddenly breaks out in the course of acute diseases coincidently with a fall of temperature. We have the *critical sweats* of pneumonia and relapsing fever; sweats which terminate a paroxysm of intermittent fever, whether of malarial or infectious origin (see fever); the profuse perspiration that attends pyemia, breaking out with each fall of temperature to disappear as it arises; the night sweats that attend tuberculosis and other exhausting diseases. In tuberculosis and in pus-formation or accumulation the oscillation of temperature, with or without chills, followed by sweating, is known as *hectic*. Sudden breaking out of general perspiration, but more notably seen on the face, attends dyspnea of pulmonary origin, and the attacks of dyspnea in the course of organic heart disease. At times sweats also occur in jaundice. In the conditions just mentioned the skin is cool and the extremities are cold.

Prolonged Perspiration.—In exhausting diseases, general and persistent perspiration may occur, particularly in the later stages, as in tuberculosis and in any disease attended by persistent dyspnea.

B. LOCAL INCREASED PERSPIRATION.—*Hyperidrosis localis* occurs when there is local vasomotor paresis. Thus in organic diseases of the brain and in affections of the peripheral nerves, in some forms of neuralgia, in migraine, and in hysteria, the symptom has been observed. Sometimes one side of the body alone is affected, even in a malarial paroxysm (hemidrosis).

Local sweats are sometimes significant. This is the case particularly with a sweat confined to the head, which occurs usually in children and is one of the striking characteristics of rachitis. The patient rolls his head at night, so that the hair on the back of the head is rubbed off.

Unilateral sweating of the head may arise from destructive pressure on the sympathetic nerves, causing paralysis of the dilator fibers of the cilio-spinal branches, in thoracic aneurism, and in caries of the lower cervical vertebra. Contraction of the pupil and congestion of the face on the same side are usually noted.

Perspiration Diminished.—*Anidrosis.*—The skin is abnormally dry in the early stages of acute disease attended by fever, particularly if the febrile rise takes place suddenly, as in acute digestive disorders of children. In adults when the disease is accompanied by high fever, as in thermic fever, the skin is dry. On the first day of the eruption of the exanthema, dryness is marked. Dryness of the skin is of frequent occurrence when there are copious discharges of water from the bowels or kidneys. In choleraic diarrhea the dryness develops suddenly. In some affections, as diabetes and Bright's disease, the dryness lasts a long time and is frequently attended by eruptions or desquamation and by the formation of boils. When there are accumulations of serum in the lymph space of the subcutaneous connective tissue, or changes in the connective tissue, as in dystrophies, myxedema, or scleroderma, the skin is dry because of the stretching and compression of the bloodvessels.

CHAPTER XIV

EXAMINATION OF THE SUBCUTANEOUS CONNECTIVE TISSUE AND THE LYMPHATIC GLANDS

SUBCUTANEOUS CONNECTIVE TISSUE

Edema.—Edema is an increase of fluid in the subcutaneous tissue. The popular term dropsy is also applied to the condition. When the condition is general it is known as anasarca. If excessive fluid is present in the larger serous cavities (the peritoneal, pericardial, or pleural) this serous effusion is known as ascites, hydropericardium or hydrothorax respectively.

The excess of fluid transudes from the capillaries and lymphatics as a result of osmosis and transudation. In the several forms of edema, not associated with an inflammatory process, various factors may play a part in causing the excess of fluid.

1. **Nephritic Edema.**—The generally accepted theory of the causation of nephritic edema assumes that there are two forms of nephritis. The first form is due to the action of circulating toxins which simultaneously causes changes in the renal and peripheral vascular apparatus resulting in nephritis and in increased permeability of the peripheral capillary walls. As a result of the nephritis there is a retention of nitrogen and of water. The retained water causes a hydremia. In turn this hydremia and the increased vascular permeability permit the escape of fluid into the tissues, causing edema. The second form is characterized by changes in the tubular structures of the kidney and decrease in the secretion of chlorides. As a result of the latter condition there is a retention of chlorides in the fluids of the tissues, causing them [the fluids] to become hypertonic. This hypertonic solution is rendered isotonic by osmosis from the blood-stream resulting in the increase of fluid in the tissues which is recognized as edema. To compensate the blood for the fluid withdrawn from it, there is a decreased excretion of water, a hydremia resulting.

It is impossible to differentiate in life between the two forms of nephritic edema, so closely are they correlated. Either form may be primary but there is soon secondarily engrafted upon it the other form, the one supplementing the action of the other.

2. **Obstruction to Venous Return or Lymphatic Outflow.**—A local or general factor may be the causative agent: if the former, pressure on a vein by enlarged glands, a gravid uterus, a tumor or an aneurism, thrombosis of a vein or obstruction to many small lymph channels,

as in elephantiasis (lymphedema); if the latter, the causative agent is found in cardiac weakness, with a consequent damming back of the blood in the peripheral circulation. In both forms transudation occurs as a result of increased pressure within the vein from the local or general obstruction, and as a result, injury to the vein endothelium from the pressure, causing increased permeability of vessel wall.

3. **Anemia.**—The edema of anemic, cachectic, and wasting conditions is usually slight. It is a result of increased permeability of the capillary wall and hydremia.

4. **Nervous Influences.**—The edema seen in neuritis, or in organic changes of the cord, is the result of nervous influences. It may be a trophoneurosis with secondary alterations in the permeability of the vessel walls, or it may be due to vasomotor paralysis, as in angio-neurotic edema.

5. **Inflammatory Edema.**—Edema, the result of inflammation, is due to an exudation of fluids into the tissues through capillaries rendered more permeable by the action upon them of the bacterial toxins elaborated at the seat of the inflammatory process. The exuded fluid possesses certain characteristic differences from the transuded fluid of a non-inflammatory edema. (See page 447.)

Mode of Recognition.—The symptoms of edema are characteristic. It changes the normal contour of the affected part; it causes deformity; its situation alters with changes in the posture of the body; it disappears temporarily to recur again; it occurs in the most dependent parts or in parts made up of loose areolar tissue; the affected part is swollen and puffy. The surface is pale, swollen, and distended. It pits on pressure, retaining the indentation for a variable time. The pitting is more pronounced if over a firm foundation, as bone. Previous edema may be recognized by the flabbiness of the overlying skin or by the striae from overstretching of the skin.

Location.—Definitely localized edema is usually of the inflammatory type. It points to underlying suppuration, and is known as inflammatory or collateral edema. It may occur over the mastoid process in mastoiditis; over the thorax in empyema; over the liver in hepatic abscess; over the joint, the seat of a purulent arthritis, etc.

Non-inflammatory edema, arising in certain parts, is a valuable diagnostic symptom. It is usually the result of pressure or thrombosis of a vein.

In the arm it is the result of pressure upon the axillary vein. If the head and arms are both edematous, then there is pressure upon the superior vena cava by a mediastinal growth. If the upper chest wall partakes of the process the pressure is below the azygos veins. Occlusion of the femoral or iliac veins causes edema of the leg; of the inferior vena cava of both legs and the lower trunk. The ascent of edema on an extremity frequently points to a thrombus gradually going up the vein.

The above forms of edema are the result of purely local changes.

When the edema results from a general cause, as in heart or kidney disease, it is usually slow in onset and affects dependent parts, although the edema of nephritis is found first in the eyes and face. The feet and ankles may be swollen as a result of circulatory changes arising from cardiac weakness either in the course of debilitating or acute infectious diseases secondarily affecting the heart or by primary diseases of the heart itself. The ankles may also be swollen in anemia, and in certain individuals of relaxed fiber after being on the feet all the day, or in hot weather. Edema of the face is typical of nephritis, and occurs most frequently in the eyelids. It differs from the edema of cardiac disease in that it is more marked in the morning. Dropsy of the scrotum and labia usually occur late in heart and kidney disease. Edema of the forehead, eyelids, or muscles is the result of trichinosis.

FIG. 19



Face of a patient with general anasarca due to chronic parenchymatous nephritis.
(Hare.)

Anasarca.—As the edema of heart and kidney disease increases it spreads from the primary seat of selection to other parts so that in a variable time the greater part of the body may become dropsical (anasarca). The anasarca of kidney disease is relatively rapid in onset. It begins in the lower eyelids, and the skin is waxy, glistening, and pale in appearance. The anasarca of cardiac disease develops slowly, commences in dependent parts and is associated with cyanosis.

Angioneurotic Edema.—This condition comes on rapidly, is circumscribed, and may involve the whole body. The attacks

may occur periodically. Local symptoms of swelling (edema) and burning and slight itching are present, and general symptoms of nausea and vomiting frequently occur. The condition may be slight, disappearing in a few hours, or may be extremely severe involving all of one side of the face; the hands may be enormous or the feet swollen to great proportions. Edema of the glottis may cause death.

Other Varieties of Edema.—*Hereditary edema* of the legs is diagnosed by the history of its presence in several generations and the absence of ordinary causes of edema. *Edema neonatorum* occurs in infants born prematurely. It is persistent, dense, and inelastic. It shows no tendency to spread. *Edema of the tongue* occurs merely with formation of vesicles, later going on to sloughing.

Diagnosis.—Non-inflammatory edema must be differentiated from (1) inflammatory swellings, exudates not transudates, by the absence of pain, heat, and redness; (2) myxedematous swellings which do not pit on pressure and which are hard and permanent; (3) connective-tissue dystrophies, which are not in dependent parts and which do not pit on pressure; (4) subcutaneous emphysema which gives a crackling sensation to the finger.

Subcutaneous Emphysema.—The rupture of an air-containing organ into the subcutaneous tissue causes either a local or general fulness of the skin. It usually occurs in close proximity to the seat of the primary trouble, which may be a malignant ulceration of the esophagus into the mediastinum, an ulceration of a tuberculous cavity when the pleura is adherent to the chest wall, or rupture of overdistended lung tissue. The swelling tends to rapid resorption or soon diffusely spreads over a large area. It gives a characteristic crepitation upon palpation. The overlying skin is pale and distended and at times tender. Subcutaneous emphysema is also produced by the bacillus of malignant edema. The tissues undergo rapid necrosis in the last condition or when there is widespread dissemination of the air in the skin.

Adiposis Dolorosa.—The occurrence of large masses of fat in the subcutaneous tissue associated with nervous phenomena, as areas of paresthesia and hyperesthesia, form a clinical syndrome known as adiposis dolorosa, or Dercum's disease. The condition is found typically in women of middle age. The cause is unknown, though it is believed in some way to be associated with atrophy of the thyroid gland. The irregularity of the distribution of the swelling and the neurotic pain distinguish the disease from ordinary obesity. (See Fig. 20.)

Connective-tissue Dystrophies are the result of a localized overgrowth of connective tissue. They occur, as a rule, on the extremities, and are firm swellings, not pitting on pressure.

Scleroderma.—The most marked characteristic of this condition is a brawny stiffening and immobility of the skin. It is either diffuse or localized, when the term *morphea* is applied to the condition. The diffuse form occurs most frequently in women between the ages of

twenty and forty. The condition usually begins with vague neuralgic pain. The skin soon becomes symmetrically indurated and hard, very

FIG. 20



Adiposis dolorosa. Note accumulations on back and on extremities. See knees and elbows; wrists and ankles unusually small. Patient, aged fifty-six years. Second attack of insanity. (Original.)

much like hard leather. The localized forms present areas of parchment-like consistency with a waxy hue. *Scleroderma neonatorum* appears from the second to tenth day after birth. The skin rapidly becomes yellowish-white, hard and stiff, and is comparable to firm cloth. Death soon ensues.

Brawny Induration.—A deep brawny induration of the legs is frequently found in scurvy. With it there is often hemorrhagic discoloration of the overlying skin. The same condition may rarely be found in syphilis.

Localized Subcutaneous Nodules.—Subcutaneous nodules may be small fatty or fibrous tumors, or both combined. They are slow of growth, painless, and the overlying skin is colorless. In fibrositis these small fibrous nodules varying in size from a small shot to a pea may be present, occurring frequently without any active symptoms. Nodules of the skin may also be tuberculous masses, gummas, sarcomas, carcinomas, or cysticercus cellulosa. The sarcoma and carcinoma nodules are at times the first manifestation of malignant disease elsewhere. Diagnosis of the tumor should be based upon the clinical history, and if in doubt the nodule should be excised and microscopic sections studied.

EXAMINATION OF THE LYMPHATIC GLANDS

Enlargement of the superficial palpable lymph glands may be indicative of a general systemic affection, or of a local malignant or infectious process in the area drained by the lymphatics running to a single gland or group of glands. Enlargement of the lymph glands in the vast majority of cases is due to a simple lymphadenitis secondary to some other condition, usually infectious in nature. *A general lymphadenitis*

may occur in any of the acute infectious diseases particularly in children and in the infectious diseases that attack them. In certain chronic diseases of children, as rickets, congenital syphilis, and tuberculosis, a general lymphadenitis is common. A general enlargement of the lymph structures throughout the body is characteristic of the condition known as the status lymphaticus (see thymus). In adults a general lymphadenitis is usual in those diseases due to animal parasites which invade the blood, notably syphilis and trypanosomiasis, and occasionally

FIG. 21



Showing location of lymphatic glands.

occurs in the acute diseases of bacterial origin. A *local lymphadenitis* is usually secondary to some local condition in the region drained by those lymphatics running to the affected gland, and is readily diagnosed. The adenitis may be acute, going on to suppuration, but more frequently is essentially chronic, the glands appearing as small discrete, painless, movable "kernels."

The following local lymphadenites are of special diagnostic significance: enlarged glands in the cervical region usually indicate some

infectious or toxic condition of the scalp, face, mouth, pharynx, or upper air passages. An enlarged left supraclavicular lymph gland is often a diagnostic manifestation of cancer of the stomach, and as with enlarged axillary glands in cancer of the breast, shows the futility of operative procedures. Enlarged epitrochlear glands usually indicate syphilis, active or latent. An inguinal adenitis is secondary to some inflammatory condition of the leg or genitalia.

Primary enlargements of the lymph glands are extremely rare. They are found in Hodgkin's disease, lymphatic leukemia at times, and lymphocythema, under which term Warthin includes all the lymphocytomatous tumors, variously known as lymphoma, lymphadenoma, lymphosarcoma, etc. All the primary enlargements of the lymph glands seem to be neoplastic in type and to run a more or less chronic course, but sooner or later terminating in death.

Tuberculosis of the Lymph Glands.—*Generalized tuberculous lymphadenitis* is rare and can only be distinguished from Hodgkin's disease by microscopic examination of an excised gland. *Local tuberculous adenitis* may be primary or secondary, and is usually the result of infection by the bovine type of tubercle bacilli. It is most frequent in young children. The glands of the neck are most frequently involved, less commonly the peribronchial and mesenteric group of glands. *Cervical tuberculous adenitis* is characterized by its extreme chronicity, the condition often coming on quickly and then persisting without perceptible change for years. The submaxillary glands are usually first invaded and the other groups later. The enlargements are hard, smooth, non-tender, at first discrete and often confluent. Both sides of the neck are usually involved, but one side more so than the other. The glands usually, sooner or later, break down with the formation of a sinus and the discharge of thick, yellowish pus. When healing takes place, disfiguring scars are left.

Peribronchial (mediastinal) tuberculous adenitis may never cause any symptoms, but if there is sufficient enlargement there may be dyspnea, cough, cyanosis, asthmatic attacks, hoarseness, etc. The x-ray will usually render a positive diagnosis of the conditions. A valuable sign in the diagnosis of the condition is the persistence of whispered pectoriloquy over the spinous processes of the vertebrae below the first, often extending down to the fifth or sixth thoracic vertebra. Impairment of resonance on either side of the upper dorsal vertebrae and over the vertebrae themselves upon mediate percussion may be elicited.

Tuberculosis of the mesenteric glands (tuberculosis mesenterica) is characterized by gradual wasting, anemia, unstable appetite, diarrhea with offensive stools, distention of the abdomen, and the presence of nodular palpable intra-abdominal growths. It is invariably fatal.

CHAPTER XV

EXAMINATION OF THE MUSCLES, BONES, AND JOINTS

THE MUSCLES

PRONOUNCED variations in the functional power and tone of muscles occurs in different normal individuals for the most part depending upon their habits of life. Variations may occur in the same individual at different times depending upon the general health, fatigue, activity, etc. Nutritional changes may cause hypertrophy or atrophy. (See also Examination of the Nervous System—the Muscles—page 192.)

Hypertrophy of the Muscles.—Hypertrophy of individual muscles occurs from overuse, as when an extremity or a portion of the trunk is used in excess. General hypertrophy of muscles occurs in Thomsen's disease. True hypertrophy is recognized by increased volume, tone, and vigor of the muscle.

Pseudohypertrophy (see under Muscular Atrophy) is associated with increased volume of muscle but diminished power.

Atrophy of the Muscles.—There are several varieties of atrophy: (1) The atrophy of disuse; (2) myopathic atrophy; (3) myelopathic atrophy, or the atrophy of degeneration, which follows lesions of the motor path, the cortex, the medulla, or the spinal cord, and develops as a result of neuritis. (See Nervous Diseases.)

The Atrophy of Disuse.—It is also known as the atrophy of inactivity. The muscles are slightly lessened in volume, soft, and flaccid. The atrophy takes place very slowly; it supervenes in certain cases of paralysis and in the joint diseases which cause immobility. It occurs also in joint disease from reflex influences. The electrical reactions of the muscles are qualitatively unchanged. By this reaction atrophy from disuse and atrophy from disease of the muscles can be distinguished from myelopathic atrophy, due to disease of the nerve (neuritis) or to degeneration of motor nerves ganglia. General atrophy of the muscles occurs in chronic cachectic conditions, starvation, and in the course of the prolonged acute infectious disease. *Myoidema* is a sudden local contraction of the muscle when it is sharply struck with the finger tip. The fasciculi rise in the little lumps which persist for a short time and gradually subside. It occurs when there is rapid atrophy of the muscles, as in phthisis.

Primary Myopathic Atrophy.—**Progressive or Idiopathic Muscular Dystrophy.**—In this form of atrophy the muscle is diseased. It diminishes in volume and finally becomes completely shrunken. In this

variety of atrophy muscular wasting takes place with or without initial hypertrophy. Three forms are seen:

1. Scapulohumeral type of Erb.
2. Facioscapulo-humeral type of Déjerine-Landouzy.
3. Pseudohypertrophic type of Duchenne. (See Diseases of the Muscles.)

Diagnostic Features of Myopathic Atrophies.—The disease is characterized by gradual progression of wasting and weakness in various groups of muscles not especially related. We never see wasting of the intrinsic muscles of the hands, as in the spinal forms of muscular atrophy, or of the tongue, pharynx, larynx, and eye. Complete paralysis rarely ensues. Electrical irritability is lessened. The reaction of degeneration is not present. Fibrillary twitchings are not seen. Sensation is not affected. The reflexes are diminished and later may be lost. The sphincters are not involved; deformities about the joints or in the spinal column may occur. The myopathies occur early in life, and are often hereditary or familial.

The *diagnosis* of idiopathic muscular dystrophy is not difficult if the above-mentioned facts are borne in mind. The fact that it occurs in family groups is an important point in the diagnosis. In cerebral atrophy there is primary loss of power. In chronic anterior poliomyelitis (spinal atrophy) wasting begins in the muscles of the hands; in both the simple and the spastic form there are reactions of degeneration, fibrillary twitching, and increase in the reflexes, and in the latter, spastic contraction of the legs.

In neuritis the paralysis is proportionately greater than the atrophy. Sensory symptoms are often present. The cause is distinct. There is no family history.

THE BONES

The Skeleton.—The bones may be the seat of pathological changes involving the entire skeleton, causing enlargement or diminution. Individual bones may be the seat of inflammation or morbid growths.

Enlargement.—Changes giving rise to enlargements of the bones occur in acromegaly, osteitis deformans, and pulmonary osteoarthropathy.

Acromegaly.—This is a skeletal change characterized by hypertrophy of the bones of the hands, feet, and face, and due to alteration in function of the hypophysis cerebri. The fibrocartilages of the ear and larynx are also enlarged. The enlargement of the inferior maxillary and frontal bones causes the face to assume a peculiar, elongated, elliptical outline. The nasal bones are enlarged and the nose thickened; the temporal fossæ are deepened on account of enlargement of the malar bones. The forehead retreats because of the enlargement of the frontal sinuses and projection of the superciliary ridges; the chin is prominent and the lower teeth project beyond the plane of the upper; the lips

and eyelids may be thickened; the tongue is enlarged and thickened. The hair is coarse and dry; the skin of the face dry and pigmented.

The hands are peculiar; they are much broader; the fingers are sausage-shaped, and the hand spade-like in form; the nails are flat, striated, and small. There is usually spinal curvature; the abdomen is prominent and the height is increased. The muscles become weak and may atrophy; the skin is often pigmented; varicose veins have been observed, and the patient complains of hemorrhoids. Hemianopsia, limitation of the visual field, and blindness or deafness may arise, due to the overgrowth of the pituitary, and other symptoms of brain tumor may occur (headache, vertigo, vomiting). Sexual changes and alterations in carbohydrate metabolism are common. In children some disorders of the hypophysis causes overgrowth without the changes of acromegaly seen in adults.

Osteitis Deformans (Paget's Disease).—There is a marked change in the contour of the patient, who exhibits a peculiar mode of locomotion. The head is advanced and lowered, so that the neck is very short, and the chin, when the head is at ease, is more than an inch below the top of the sternum. The chest becomes contracted, narrow, flattened laterally, deep from before backward, and the movements of the ribs and spine are lessened; the arms appear unnaturally long; the shafts of each tibia and femur are bent so that the patient becomes bow-legged. There is some stiffness, but no loss of power and not a great deal of pain. The skull is increased considerably in thickness.

These changes cause a dwarfed appearance of the trunk in comparison with the legs and arms, and the posterior lateral curvature necessitates a characteristic attitude. The skeletal changes are noted particularly in the long bones. As a result of the enlargement of the cranial bones the face presents a triangular outline, with the base above and the apex below (see Fig. 23, outline 3), thus differing in appearance from the outline of acromegaly (see Fig. 23, outline 2).

FIG. 22



Case of acromegaly. (Osborne.)

Pulmonary Osteo-arthropathy.—There is hypertrophy of the bones of the extremities, including enlargement of the shafts. The bones of the face and head are not affected. The hands and feet are enlarged, and the patellæ and other bones of the knee-joints increased in size.

FIG. 23



FIG. 24



Pulmonary osteo-arthropathy. Female, aged eleven years. Tuberculous vertebral caries and pulmonary tuberculosis. Enlarged clubbed fingers and thickened ulna and radius. Private patient, 1885. (Original.)

Curvature of the spine is present. The appearance of the fingers is different from that seen in acromegaly. The ends are enlarged and bulbous, and the nails are large and curved both in the transverse and in the longitudinal direction, like the clubbed fingers of phthisis; although

the chief enlargement of the fingers is not terminal, and there is no cyanosis as in phthisical clubbing. The change seemed to be associated with pulmonary affections, and Marie called it *osteo-arthropathie pneumonique*.

Diminution.—Imperfect development of the bones is seen in idiots and cretins; diminution in size of the skeleton with deformities may occur from rickets (*q. v.*) or from osteomalacia.

Osteomalacia.—The bones are soft, break on the slightest provocation, or bend in various directions, depending upon the external pressure and the direction of the muscular force. The ribs are drawn in by the inspiratory force until the cavity of the thorax is lessened to a degree incompatible with life. The pelvis is deformed so that labor is impossible. (The disease frequently develops during pregnancy.) All sorts of fixed contortions are assumed. If the patient is able to be up the body shortens, the back becomes rounded, and the neck flexed so that the chin is brought close to the sternum. The bones can be indented with the finger, and crackle like egg-shells.

Nodules or Nodes.—Bone swellings are usually due to periostitis or necrosis, the causes of which are trauma, infection, and syphilis. In syphilitic periostitis the swellings form on various portions of the skeleton, but are seen most frequently on the skull, especially on the forehead. They are also found on the ribs, the sternum, and the shafts of the long bones, preferably the tibia, ulna, and clavicles. They are usually multiple or bilateral. The overlying skin may be the seat of edema. They are painful and tender on pressure, and may be the seat of heat and redness. Periostitis is also seen in scurvy and rickets when the gums are affected. Exostoses are hard and dense. They are situated on the outer aspects of the bone and in relation to the strongest tendons or muscles.

Tenderness of Bones.—Tenderness upon pressure upon the long bones is an occasional symptom in general disease as syphilis and in diseases in which the bone-marrow is probably affected as pernicious anemia and leukemia. Localized areas of tenderness with fever at once suggest some acute inflammatory bone disease (epiphysitis, periostitis, and osteomyelitis). Deep-seated pain is indicative of osteomyelitis. It is common in multiple myeloma, a disease characterized by bony enlargements, a tendency to spontaneous fracture, various sensory symptoms from the local growths, fever, anemia, and the pathognomonic sign of Bence-Jones albumosuria.

Cervical Rib.—An accessory cervical rib may be the cause of pain and paresthesia in the arm and hand. The rib may or may not be palpable, but an x-ray examination will always disclose it.

The Spinal Column.—No physical examination is complete without an investigation of the movability of the spines and the presence or absence of curvature. Modifications of the position and movements of the spinal column are due to muscular weakness or paralyses (diphtheria, etc.) and to arthritis and caries. The symptoms of

arthritis are localized tenderness, pain, muscle spasm, and rigidity. Tenderness of the vertebræ may be brought out by percussion and by pressing on the patient's shoulders while he is standing upright. Percussion may cause pain in meningitis and myelitis. The passage of a sponge, wrung out of warm water, down the spine may cause pain over the site of disease in myelitis. Tumors of the vertebra may be detected by palpation anteriorly and posteriorly. Pain in the distribution of the nerves or at their termination is often due to spinal caries with pressure on them as they pass through the foramina. The most notable is the pain about the umbilicus due to Pott's disease in children. These conditions cause curvatures which may be lateral or anteroposterior.

Curvatures.—Lateral curvature (scoliosis) is due to muscular weakness, deformities of the chest, hip disease, etc.; anteroposterior curvature (kyphosis) to rickets, old age, occupations, acromegaly, emphysema, Pott's disease. An exaggeration of the normal lumbar curve (lordosis) occurs in abdominal tumors (notably pregnancy and ascites), progressive muscular atrophy, pseudohypertrophic dystrophy, cretinism, and congenital dislocation of both hips.

Disorders of the gastro-intestinal tract and the uterus are undoubtedly intensified by the presence of curvature, which leads to deformity of the body, and hence to the assuming of abnormal positions while sitting or walking. The recognition of lateral or anterior curvature leads to the adoption of lines of treatment which otherwise would not be followed, but without which weak muscles, improper aeration of the blood, and sluggish circulation would persist.

THE JOINTS

The size, shape, and color, the degree of mobility, and the position of the joints are observed. The number of joints involved and the duration of the process in each joint must be carefully noted.

Size and Shape.—1. In enlargement due to infiltration about the joint the tissues are thickened, as shown by palpation, and the outline of the joint is changed. The normal contour is lost entirely, and instead there is a globular swelling beginning above and extending below the joint.

2. Enlargement, if due to effusion, may be detected by palpation, as this elicits fluctuation, or by aspiration. This is particularly so in the large joints. If the joint involved is the knee, the patella will float. The effusion changes the normal contour, but may in the earlier stages cause local swellings where the synovial sacs are near the surface; hence, at the articulation of the tibia and fibula with the tarsus, on the inner and outer side, a boggy swelling is observed. At the knee the swelling is on each side above and below the patella. When the effusion is great the joint becomes immobile and may be flexed from distention of the sac.

3. When enlargement of the joints is due to hypertrophy of the bones, as in Charcot's disease, the latter are thickened and very hard. There may or may not be, and usually is not, fixation, and movement is but moderately interfered with.

4. Atrophy of the muscles causes a pseudodiminution in size of the joint. Changes in the outline of the joint are also seen in arthritis deformans. The loss of the cartilaginous substance of the joint, with the secondary osteophytic changes, causes deformity, so that in the case of the small joints of the finger subluxation is seen; similar subluxations are seen in larger joints. The ends of the phalangeal bones are thickened.

Color and Position.—Change in color is usually noticed in inflammations. The surface is either bright red or dusky. The position assumed is of diagnostic importance. Flexion of the limb of the affected joint occurs in overdistention. It must be remembered that the hip-joint is flexed in appendicitis and in psoas abscess or other affections in proximity to the psoas muscles.

Palpation.—By palpation we determine the degree of mobility, employing passive motion of the joints; the presence of fluctuation and of crepitation; the condition of the bones, if thickened or irregular; the presence of bulging or boggiess along the margin of the joint from thickened synovial membrane.

1. The *mobility* of the joint is lessened or wanting because of pain, muscular spasm, distention of the capsule by effusion, the presence of osteophytes, and ankylosis. Movement is merely inhibited in inflammation on account of the pain. Spasm prevents movement. A reflex muscular spasm takes place if osteitis and cartilage destruction are present.

2. *Fluctuation* is revealed by palpation and points to liquid effusion within the joint. Edema of the surrounding tissues occurs with purulent effusions.

3. A *crepitus* or grating sensation is observed in arthritis deformans and other destructive diseases. It may be heard as well as felt.

4. *Aspiration*. The fluid removed must be examined by bacteriological methods.

Joint Lesions.—Joint lesions or processes may be expressions of general infections, as septicemia, influenza, pneumonia, cerebrospinal meningitis, scarlet fever, and dysentery; of blood diseases, like purpura, hemophilia, or scurvy; nervous diseases, like tabes dorsalis, or of metabolic diseases, as gout.

We have to consider synovitis or arthritis, single and multiple, traumatic or infectious, gonorrheal and tuberculous infections being the most common monarticular causes. We shall then consider gout, arthritis deformans, and finally the neuropathic joints.

Acute Synovitis and Arthritis.—The inflammation is recognized by pain, heat, redness, and swelling. Effusion is present and its physical signs are readily elicited. It is both periarticular and intra-articular.

It may be due to traumatism; but we are now chiefly concerned with inflammations due to internal morbid processes. When single joints are affected, the most common causes are tuberculosis, pyemia, and gonorrheal infection. When many joints are affected, the cause is an infectious one, as acute articular rheumatism, septicemia, pyemia, epidemic cerebrospinal meningitis, scarlet fever, and dysentery, rarely gonorrhea.

Tuberculous Arthritis.—In tuberculosis the joint is swollen and the neighboring tissue edematous (white swelling). Effusion may be detected. The hip, the knee, the elbow, the wrist, and the ankle are most frequently affected. Destruction ultimately takes place, with subluxation and subsequent fixation of the joint. With fever, wasting, and local signs of tuberculosis in other portions of the body the tuberculous nature of the affection is indicated.

Gonorrheal Arthritis.—One of the larger joints is usually affected. Signs of acute or subacute inflammation are present, with edema and effusion. The patient is a male in whom an acute or chronic urethral discharge is found. The pain is worse at night. The process is of long duration. The general and local signs of acute articular rheumatism are wanting. In certain cases several joints are affected, but the temperature is not so high nor the sweats so profuse as in acute rheumatism.

Gout.—The acute joint, usually the metatarsal phalangeal articulation of the big toe, is red, hot, tender, and swollen. Repeated attacks cause great deformity. Periarticular tophi form and frequently ulcerate through the skin.

Arthritis Deformans.—Three forms are seen: (1) Atrophic arthritis: In the early stages the joints are usually acutely inflamed and swollen. Repeated attacks result in pronounced deformities, usually more marked in the smaller joints and gradually involving the larger ones. (2) Hypertrophic arthritis: The joint is somewhat painful and more or less disabled. There is hypertrophy of the cartilages along the outer margin of the joint, which becomes ossified. In the fingers, Heberden's nodules and in the back spondylitis deformans exemplify two types of the condition. (3) Chronic villous arthritis: The joint, usually a large joint, is slightly swollen, soft, and painful and creaks on motion.

Arthritis in Hemorrhagic Diseases.—In hemophilia and purpura there may be in the larger joints acute inflammatory processes resembling acute articular rheumatism. In scurvy there is frequently an arthritis occurring as a complication.

Neuropathic Arthritides.—In forms of nervous disease, particularly in sclerosis of the posterior columns, secondary joint involvement sometimes occurs. The change in the large joints is preceded by stiffness and disability. Gradually trophic changes take place. The cartilages become eroded, the heads of the bone waste, the ligaments ossify, and irregular bony growths project. Wasting of the head of the femur is followed by dislocation. Sometimes an effusion takes place into the joint, and there may be periarticular edema. The large

joints, knee, hip, ankle, and elbow, are most commonly affected. When the tarsal bones and the articulations are affected the foot becomes flat, and the tarsal and metatarsal articulations and the tarsal bones project forward to backward. This is called the tabetic foot.

Symptoms of joint diseases are seen in hysteria. Pain and fixation of the joint are sometimes present. The joint rarely undergoes organic changes, but sometimes a plastic infiltration of the connective tissue outside of the capsule does occur. The hysterical nature of the pain and immobility are recognized by the absence of a cause for joint lesion, the absence of fluctuation or of signs due to erosion, by the association of the local symptoms with the phenomena of hysteria, but more particularly by the fact that contraction and even wasting precede the joint symptoms. In true affections of the joint both occur after the joint has become diseased. In hysteria muscular contraction takes place first.

CHAPTER XVI

EXAMINATION OF THE UPPER RESPIRATORY AND ALIMENTARY TRACTS

THE NOSE

The Exterior.—The shape of the nose is changed in myxedema (*q. v.*) and in disease of the bone due to syphilis. The latter causes sinking or depression of the bridge of the nose, which must not be confounded with the depression that occurs in fracture. The nose may be broadened in cases of tumors of an expanding nature in the nasal cavities. The nose is the seat of eruptions as acne and of hyperemia, but they are usually of local origin.

The Interior.—Examination of the cavities of the nose consists of two procedures. These are anterior and posterior rhinoscopy.

1. **Anterior Rhinoscopy.**—For this are needed a Welsbach or Argand light, a nose speculum of some form, probes, a 5 per cent. solution of cocaine, and a head mirror with central opening.

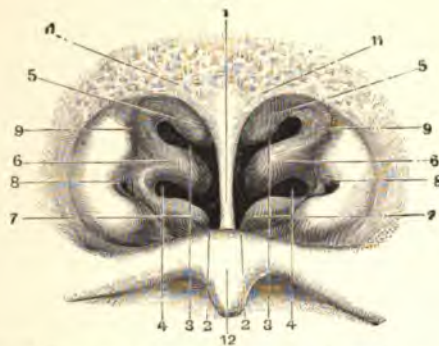
The patient is seated facing the surgeon, with the light behind and at the side of the ear, as nearly as possible on a level with the examiner's eye. The central aperture of the head mirror is in front of the operator's eye, and the reflected light falls on the patient's nose. The speculum is then taken in one hand and the nostrils dilated, so that the view of the interior is unobstructed. Proceed from before backward with the examination, carefully focussing the light on each part in succession and gradually tilting the head of the patient backward. Thus the floor of the nose, the septum, inferior turbinated bone, middle turbinated bone and sometimes the superior turbinated bone, are brought into view successively. In a broad nose one may at times see the posterior wall of the pharynx, which is distinguished by its peculiar wave-like movement when the patient swallows.

With the probe the operator tries the condition of the mucous membrane, tests the consistency of tumors or hypertrophies, and so judges the character of the condition. After this the enlarged parts should be touched with cocaine and the result observed. Contraction of a swelling under its influence proves its vascular origin.

2. **Posterior Rhinoscopy.**—The instruments needed are a tongue depressor, head reflector, two sizes of throat mirrors, a palate hook, and a curved applicator for cocaine, or a spray bottle with tip turned upward. The tongue is held down by the tongue depressor, and the

patient is told to breathe freely through both mouth and nose. The light is directed into the pharynx and a mirror of the largest possible size inserted carefully behind the soft palate. The proper angle and the movement necessary to bring all parts into view can only be learned by practice. As a rule it is always best to hold the handle well up at first and note the condition of the vault of the pharynx, then gradually depress it, examining the choanæ from above downward. After the choanæ have been examined a turn of the mirror to either side will bring into view the orifices of the Eustachian tubes, and the examination is complete. If it is impossible to see the posterior nares, it may be necessary to resort to the palate hook to hold the uvula forward, applying cocaine to the soft palate before inserting it.

FIG. 25



Rhinoscopic image. 1. Vomer or nasal septum. 2. Floor of nose. 3. Superior meatus. 4. Middle meatus. 5. Superior turbinated bone. 6. Middle turbinated bone. 7. Inferior turbinated bone. 8. Pharyngeal orifice of Eustachian tube. 9. Upper portion of Rosenmüller's groove. 11. Granular tissue at anterior portion of vault of pharynx. 12. Posterior surface of velum. (Seiler.)

By the above methods the appearance and nutrition of the mucous membrane, relative size of the cavities, the nature of the discharge, and the presence of ulceration or perforation of the septum are determined. Deviations of septum, enlargement or contraction of turbinated bones, the size of the cavities, and the presence of foreign bodies or abnormal growths are also detected.

Appearance of the Mucous Membrane.—Pallor is seen in tuberculosis and in atrophic rhinitis. If a protuberant mass is observed to be transparent and shining, as well as pale, it is a *polypus*. If the mucous membrane is bright red, it may be due to *acute inflammation*, to *glanders*, or to *syphilis*. It is dull red in *chronic catarrhs* and *caseous rhinitis*. The coatings of the mucous membrane are of significance. If a dry mucus covers the part, there is *dry catarrh*; on the other hand, a dirty gray membrane is indicative of *diphtheritic rhinitis*.

^a *chronic hypertrophic rhinitis* the uvula is thickened and elongated.

The *anterior margins of the edges of the turbinated bones are enlarged throughout or in location.* The mucous membrane covering these *apex is thickened, hard, and rough, not contracting when touched with a probe.* The *posterior ends of the inferior or middle turbinated bones are continuously enlarged, forming round tumors which obstruct more or less the posterior nares and project into the pharynx; polyps and prolapsion of the septum may complicate these cases.* The same appearance is seen in chronic postnasal catarrh, and in addition a *granulated and thickened appearance of the pharyngeal mucous membrane and that of the posterior third of the septum.* In dry catarrh the mucous membrane is thin, pale, hard to the touch, and covered with a layer of dried secretions and crusts, and one or all three turbinated bones are enlarged.

Nasal polyps are grayish-yellow or greenish, shiny masses, on a broad base, soft and yielding to the probe; they are usually not circumscript.

Ulceration of the mucous membrane is usually a manifestation of lupus, tuberculosis, or tertiary syphilis. In *lupus* the ulceration has extended from the exterior. The ulcers may be followed by *necrosis and caries of the bones.* If the *ozena* is not removable by antiseptic sprays the bones are probably affected. A discharge of *sequestra* makes the diagnosis positive. Tuberculous ulcers are usually found in the septum. They are rarely primary. They present a whitish-gray surface, with elevations of infiltrated tissue. They bleed on the slightest provocation. In *syphilis* the ulcers are situated anywhere in the nares; the stench of the breath is sickening, and the patient complains of stomatitis and loss of smell. There may be some localized tenderness. There may be more superficial excoriations, or deep serpiginous ulcers, surrounded by an inflammatory zone. Caries can be detected with a probe. The ulcerated surfaces are covered with a dry, greenish crust. Foreign bodies usually cause ulceration if impacted.

Trophic ulcers are painless and spread rapidly over considerable surface; they follow paralysis of the fifth nerve. They are dry and sloughy and do not extend to the skin. *Postfebrile ulcers* follow measles, scarlatina, typhoid, and variola, and are due to rupture of small abscesses with the subsequent formation of ulcers. They are usually situated anteriorly on the septum or on the inner side of the alæ, covered with scabs and very irritable.

Perforation of the septum frequently occurs in the anterior and lower part of the cartilaginous portion.

Nasal Secretion. The odor of the nasal discharge may be suggestive of dystheria or of the presence of foreign bodies. The discharge in the latter instance is sanious or purulent. Animal parasites, as well as peas and beans, cause pain, symptoms of obstruction, and ulceration. In *syphilis* with caries the odor is usually gangrenous.



PLATE II.

MODIFIED FROM SCHNITZLER'S ATLAS.

Fig. 1. The normal larynx as it appears during inspiration.

Fig. 2. The normal larynx as it appears during phonation.

a. Epiglottis.

b. Left ventricular band.

c. Left vocal cord.

d. Eminence marking the site of the left cartilage of Wrisberg in the ary-epiglottic fold.

e. Eminence marking the site of the left cartilage of Santorini and practically also that of the arytenoid cartilage in the ary-epiglottic fold.

f. Interarytenoid space with a slight amount of the upper portion of the posterior wall of the larynx visible.

g. Trachea with its rings.

h. Right ventricle of the larynx.

Fig. 3. The larynx in a moderate attack of acute laryngitis.

Fig. 4. Early stage of tubercular laryngitis. Localized hyperæmia and thickening on the posterior portions of the vocal cords. Infiltration of the posterior wall of the larynx with tubercle formation. Pale, cedematous swelling of both ary-epiglottic folds obliterating the eminences of Wrisberg. Anæmia of the laryngeal mucous membrane.

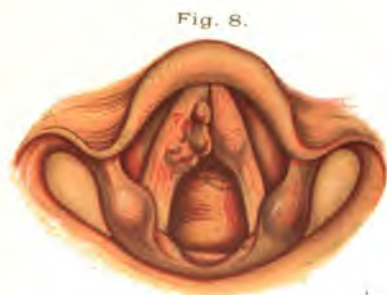
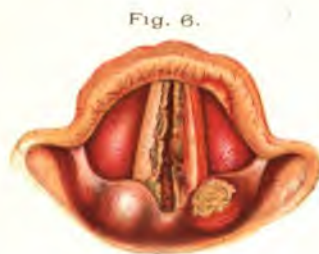
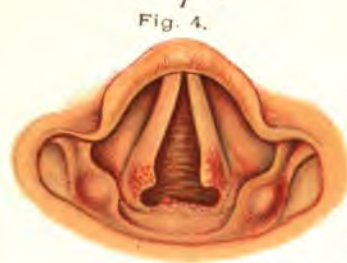
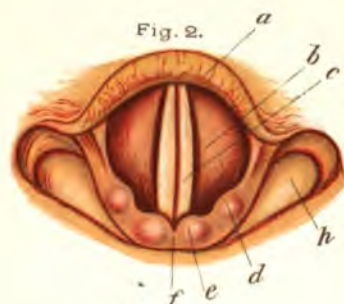
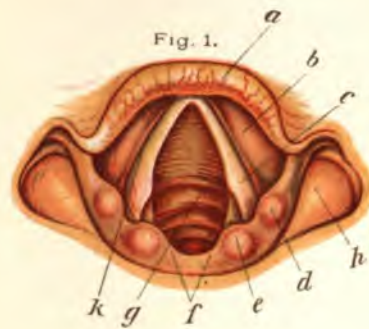
Fig. 5. Large, lobulated, tubercular infiltration on the posterior wall of the larynx. Commencing ulceration at the most prominent part of the growth.

Fig. 6. Tertiary syphilitic ulcer of the left ary-epiglottic fold and margins of the vocal cords. The right ary-epiglottic fold is the seat of a gumma that has begun to soften. The under portion of the epiglottis on the left side is superficially ulcerated from contact with the secretions of the ulcer on the ary-epiglottic folds.

Fig. 7. A papilloma attached by a broad base to the left vocal cord. It extends across the glottis and rests upon the right vocal cord. Owing to the weight of the tumor the action of the left crico-arytenoideus posticus is impaired and the left vocal cord is straighter and abducted less than the right one.

Fig. 8. An epithelioma involving both vocal cords. The left is merely infiltrated, while the right is ulcerated slightly. The deep red or purplish color of the passive hyperæmia should be contrasted with the appearance of the mucous membrane in other inflammatory conditions.

PLATE II



THE LARYNX

The objective symptoms are determined by inspection and palpation. Inspection of the exterior of the larynx reveals the presence of swelling, and the movements of the organ as a whole. Local swelling of the tissues over the larynx may occur in inflammations of the cartilages, which are usually of syphilitic origin, but may attend carcinoma or other tumors. More or less marked swelling attends the inflammation of the cartilages; fluctuation after a time is detected, and when the abscess is opened, pus and necrosed cartilage are evacuated. The objective signs of inflammation are noted.

The movement of the larynx is increased in cases of dyspnea. It is accompanied by recession of the spaces above the sternum and the clavicles, with clonic contraction of the sternocleidomastoid muscle. The interior of the larynx is studied by inspection (laryngoscopy) and by palpation (probe or fingers).

Examination.—The same instruments are used as for posterior rhinoscopy. The patient is seated as for the rhinoscopic examination. The laryngeal mirror should be moderately heated, so that when it is placed in the mouth the vapor of the breath will not be condensed on its surface. The patient must open the mouth and protrude the tongue, which is grasped gently but firmly between the folds of a napkin by the thumb and fingers of the examiner. The mirror is then inserted carefully and quickly, face downward, into the pharynx. Care must be taken not to touch the tongue or palate, otherwise the patient may retch and become alarmed. The mirror is passed to the posterior wall of the pharynx, and so directed that the image of the larynx is reflected to the eye of the operator. The patient is made to pronounce "a" or "ee," not "ah," and then to respire. The various structures and the action of the cords are observed. The appearance of the mucous membrane is studied during quiet respiration.

The epiglottis is very dependent, so that often the larynx can only be seen by having the patient stand while the operator remains seated. The patient's head is bowed on his chest and the examination proceeds.

The first examination may not result satisfactorily, but little being observed on account of spasm of the pharyngeal muscles. Repeated sittings may allay apprehension and accustom the mucous membrane to the presence of the mirror. This object may be attained by applying cocaine to the pharynx.

The probe is needed only to ascertain the consistency of tumors and growths. Cocaine may be applied before it is used.

The Appearance of the Larynx in Health.—Fig. 1 and 2 of Plate II show the larynx as it is seen in the laryngoscopic mirror. Above (upper part) is the arched epiglottis, below is the cavity of the larynx. In the centre are the vocal cords, white and glistening; on each side the pink folds of the false cords. At the bottom of the mirror

are the arytenoid bodies, and between them the folds of the interarytenoid space. Below and outside the arytenoid bodies are the fossæ. The mucous membrane is pink throughout except on the cords. In respiration the arytenoids separate, carrying the ends of the cords, which are attached to them, with them, and leaving a triangular opening—the glottis—through which the rings of the trachea can be seen. In phonation the arytenoids approach each other, obliterating the interarytenoid space: the inner edges of the cords come in contact and close the glottis. (See Plate II, Fig. 2.)

Appearance in Diseases.—A note must be made of the color of the various parts, of the presence or absence of swellings, of ulceration, of new growths, and of alterations of the movements of the parts concerned in phonation, particularly of the cartilages and the cords.

Color.—The color is an indication of the degree of congestion. *Anemia* of the larynx may be merely part of a general anemia from any cause. An intense anemia of the larynx is an early and valuable sign of pulmonary tuberculosis.

Hyperemia may be active or passive. It is readily recognized by the intense redness. Active hyperemia occurs in acute laryngitis, either of the primary or secondary forms. Passive hyperemia occurs in general obstructions to the circulation, as emphysema or valvular lesions, pressure on veins by tumors, etc. Active hyperemia leads to catarrh, passive to edema.

Swelling and Infiltration.—Swelling of the epiglottis and of the aryteno-epiglottidean folds is seen in edematous laryngitis, in acute, subacute, and chronic laryngitis. In *edema* of the glottis the swelling is below the vocal cords. The swelling may be circumscribed and undergo suppuration. Swelling and edema are also seen in *perichondritis*.

TUBERCULOSIS.—Swelling and infiltration succeed the primary anemia or catarrh of the first stage of laryngeal tuberculosis. At first there are slight intumescences of tuberculous infiltration, not well outlined, and gray in color. They are found most frequently in the interarytenoid space, less often on the false cords and arytenoid cartilages, rarely on the epiglottis.

1. A hill-like prominence between the arytenoid cartilages either in the middle or on one side. During phonation it comes up between the cords.

2. When a false cord is affected, the whole of it is usually infiltrated, forming a tumor-like swelling which often hides the vocal cords.

3. *Vocal Cords.*—At first usually only one cord is affected. It is thickened and the free border is red. Sometimes the free edge seems split. The infiltration may extend to the subcordal region and cause a hypoglottic laryngitis.

4. *Epiglottis.*—Infiltration of the epiglottis is rarer than edema after ulceration, and care must be taken not to confound these conditions. The whole epiglottis, or only portions of it, may be affected. The structure thickened and curled upon itself, and not freely movable.

5. *Arytenoid Cartilages*.—They appear enlarged and puffy, and often fixed from perichondritis. Thickening of either one or both of the arytenoid cartilages is most characteristic.

SYPHILIS.—In syphilis we have three forms of swelling: 1. *Mucous Patches*.—These are transient flat elevations of 3 to 7 mm. diameter, oval or circular, and of a whitish-gray color. When the epithelium is lost they appear yellow and purulent.

2. *Infiltrations*.—They are diffuse thickenings in various parts of the larynx, most often on the epiglottis, which may be thickened uniformly or only in part around the edge. The vocal cords may be so swollen as to cause dyspnea. Usually an ulcerated spot is seen in the centre of the infiltration. The mucous membrane is either normal or reddened.

3. *Gummas*.—Gummas appear as round prominences of the same color as the surrounding tissue. They occur on either side of the epiglottis, on the aryteno-epiglottic folds, often in the interarytenoid space, on the false cords, and on the under surface of the vocal cords. If they break down, deep ulcers form, leading to extensive destruction of the parts.

LUPUS.—In lupus isolated or grouped nodes are seen coalescing into patches on the epiglottis. The disease is usually present on the face or in the pharynx and mouth.

FISSURES.—Fissures and erosions are present in chronic laryngitis.

ULCERS.—Ulceration is seen in tuberculosis, syphilis, carcinoma, leprosy, and lupus.

Tuberculosis.—Ulceration occurs in tuberculosis in the following situations: (1) Interarytenoid space. (2) False cords. (3) Aryteno-epiglottic ligaments. (4) Vocal cords. (5) Epiglottis (laryngeal surface). The ulcerations are usually flat and superficial, though at times destructive.

Syphilis.—Syphilitic ulcers are circular, deep, with a sharp border and inflammatory areola, and overlaid with a whitish-yellow deposit. They develop from an infiltration or a gumma, and not on an unchanged surface. Ulcers on the upper surface of the epiglottis are always syphilitic.

TUMORS.—**BENIGN TUMORS**.—*Papilloma*.—Three varieties are met with: (1) Small warty growths, usually on the cords, dark red in color, and seldom larger than a bean. (2) Groups of raised white papillæ on a broad base, also growing on the cords. (3) Large red, mulberry-shaped growths, partly villous, partly warty, which fill up the whole larynx.

Fibroma.—Fibroma appears as a hemispherical, pedunculated tumor of dirty white, reddish, or dark red color, more or less dense in consistency. It is usually single, and grows most frequently from the cords. The smallest examples are known as "singers' nodes;" they may be as large as a hazelnut.

MALIGNANT TUMORS.—*Carcinoma*.—*Epithelioma* (the most common form) is seen as a circumscribed, hemispherical, warty, or cauliflower-

like formation, varying in size, or as a knotty infiltration projecting into the larynx. The *medullary* form is larger, soft, and bloody, and rapidly ulcerates. *Scirrhous* is firm and hard. The structure of the larynx is gradually invaded and necrosis of the tissues develops. Perichondritis and abscess frequently ensue.

In carcinoma of the cords two kinds of growth are seen. In the *polypoid* form the tumor develops on the cord like a warty growth, sometimes papillary and of a reddish-gray color. In *diffused* cancer of the cord the structures are red and knotty, and invade the surrounding tissue without distinct demarcation.

Sarcoma.—The tumor (rare) has a broad base, is shiny in appearance, and sometimes lobulated. Sometimes the structure is dark red or yellow.

Sputum.—The sputum from the larynx is generally scanty; it is not frothy and is colorless and transparent; it is often discharged in small globules; it may be streaked with blood. Sometimes pseudomembranes are coughed up. It is doubtful if purulent sputum ever comes from the larynx, excepting in cases of perichondritis, in which the abscess bursts into the larynx. Laryngeal sputum is found in catarrh and malignant tumors. It is blood-streaked when the catarrh is very intense or after injuries.

The Epiglottis.—The epiglottis when inflamed is swollen and red, and may be palpated with the finger.

THE MOUTH

Color.—Pallor is associated with anemia. Increased redness attends inflammation. The mucous membrane is yellow in jaundice, bluish in cyanosis, best seen under the tongue. The mucous membrane is the seat of pigmentation in Addison's disease and in argyria. In the former, small oval, purplish spots are seen, and must not be confounded with the pigmented spots common after stomatitis in negroes. Eruptions occur in the mouth, and may precede the cutaneous eruption. This is notably so in measles. Vesicles are seen in smallpox and chickenpox.

Shape.—The floor of the mouth is encroached upon by enlarged glands underneath or by swelling of the cellular tissue. Bone diseases and some teeth affections cause swellings. The dental arch is narrow and high in adenoids or from "thumb-sucking."

Fetor.—The odor may be a simple fetor or of a metallic or gangrenous character. Fetor attends all inflammations; it is more pronounced in ulcerative and mercurial stomatitis; in the latter it may be metallic.

Hemorrhage.—Petechiæ are seen in purpura hæmorrhagica; submucous hemorrhages in scorbutus and severe forms of purpura are common on the cheeks and on the gums. In ulcerative endocarditis hemorrhagic infarcts may be seen; in grave anemias petechiæ are also seen.

Capillary oozing of blood takes place from the mucous membranes in severe infections. The accumulated blood collects about the teeth and on the tongue, and in febrile states becomes dry. Dry incrustations are known as *sordes*.

Salivation.—Increased flow of saliva occurs in all inflammations of the mouth unless attended by high fever. It may be voluntarily discharged by the patient or dribble in a continuous stream. (See Saliva.)

The Gums.—The gums may be the seat of inflammation and ulceration, particularly in certain metallic poisonings. The gums swell and grow spongy. A red line at the junction of the gums and the teeth, the *gingival line*, is frequently seen in cases of tuberculosis, and often in other cachectic conditions, as carcinoma, and in diabetes.

In *scurey*, the gums are swollen and spongy. They bleed easily, and are usually streaked with blood. Ulcers form along the alveolar margin. The gums of decayed teeth are usually the seat of inflammation.

A *blue line* is seen at the margin of the gums in lead-poisoning. Before the line makes its appearance, a row of discrete black dots occupying the seat of the papillæ of the mucous membrane is observed. If examined with a magnifying glass, the line is readily seen to be an interrupted one. It does not always extend along the entire margin, but may be limited to a few front teeth in either the upper or the lower jaw. It can be distinguished from dirt on the teeth by placing the corner of a small piece of paper under the edge of the gum.

The Teeth.—Cases of indigestion, chronic gastritis, constipation, and diarrhea are often due to defective mastication. Persistent aural, nasal, and ophthalmic affections may have their primary origin in disease of the teeth. Caries of the teeth may cause headaches or neuralgias, near or remote, and may explain many cases of foul breath.

Pitting of the surface of the teeth and thinning of the enamel in transverse grooves are held by some to be due to mercury, by others to infantile stomatitis. The color of such teeth may be darker than normal. A transverse furrow crosses all the teeth at the same level. *Erosion* of the teeth takes place in gouty subjects. There are wasting and loss of polish of the labial surface, followed by deep grooves which extend into the body of the teeth. In *pyorrhea alveolaris* there are usually marginal inflammation of the gums, inflammation and necrosis of the pericementum, loosening of the teeth, and formation of so-called calculi.

Hutchinson's Teeth.—In cases of *congenital syphilis* the upper central incisors of the permanent set are affected. They are dwarfed, narrowed, and short. The middle lobe of the tooth is atrophied so as to have a single broad vertical notch in the edge of the tooth. A narrow furrow sometimes passes upward from the notch on both the anterior and the posterior surface nearly to the gum.

Teething.—Many authorities believe that the eruption of the teeth takes place without the occurrence of general or reflex symptoms,

equally careful observers believe that the phenomena of the feverishness, insomnia, etc., often attend the process.

FIG. 26



Notched teeth. Malformation of permanent teeth found in hereditary syphilis.
(Jonathan Hutchinson.)

Slowness in the development of the teeth may be due to rachitis. The student should be familiar with the periods of development and the number of teeth that appear at each period.

DATES OF ERUPTION OF THE TEETH

Milk Teeth

2 M	1 C	4 I	1 C	2 M	
					=20
2 M	1 C	4 I	1 C	2 M	

Eruption of central incisors about	7th month ¹
Eruption of lateral incisors about	9th month
Eruption of first molars about	15th month
Eruption of canines about	18th month
Eruption of second molars about	24th month

Permanent Teeth

3 M	2 B	1 C	4 I	1 C	2 B	3 M	
							=32
3 M	2 B	1 C	4 I	1 C	2 B	3 M	

Eruption of anterior molars about	7th year
Eruption of central incisors about	8th year
Eruption of lateral incisors about	9th year
Eruption of anterior bicuspid about	10th year
Eruption of posterior bicuspid about	11th year
Eruption of canines about	11th year
Eruption of second molars about	12th to 14th year
Eruption of third molars (wisdom teeth) about	18th to 25th year

The Tongue.—Moisture.—The moisture is due to the saliva, any deficiency of which causes dryness of the tongue. Excess of salivary secretion has no direct effect upon the tongue.

Dryness may be due to increase of evaporation from keeping the mouth open, as well as to diminution of the salivary secretion. In chronic fevers the dryness is due to the effects of the temperature upon the secretions in general, but it is not the effect of high temperature, curiously, but rather a temperature which has persisted for a considerable length of time. Thus in pneumonia with a temperature

¹ Lower incisors first.

of 105° the tongue may be moist; whereas in typhoid fever with a temperature of 103° the tongue is dry. General dehydration of the body causes dryness of the tongue even without local diminution of secretion, hence it is a common symptom in diabetes, after severe hemorrhages, in disease associated with severe diarrhea and at times after severe sweats. It is curious to observe that in cholera the tongue remains moist even until death; whereas if the patient is about to improve and the discharge ceases, reaction and fever setting in, the tongue begins to dry and becomes quite brown. Prostration and asthenia in all forms continuing over a moderate period of time, as a week or ten days, causes lingual dryness. In *xerostomia* a condition occurring in neurotic women, characterized by deficiency or actual arrest of salivary secretion, the tongue is dry, fissured, and red.

Color.—The tongue is pale in anemia; red in inflammatory conditions of the mouth and in fever; yellow in jaundice; and purplish in cyanosis.

Pigmentations.—Yellowish-white oblong patches, soft and slightly raised, are sometimes seen along the sides of the tongue, upon the eyelids, and upon the palms of the hands, rarely in other portions of the body—*xanthelasma*. Dark purple, bluish-black or black marks are seen after glossitis on the tongue as well as on the surface of the lips; the latter are sometimes the seat of brown pigmentation. The macules are sharply defined, neither raised nor depressed, and vary in size. Other pigmented areas are also found in Addison's disease. Blood-stains are observed in purpura. Submucous hemorrhages or ecchymoses are seen in anemia, in purpura, and other hemorrhagic states. Hemorrhagic infarcts are seen on the tip.

In *nigrities*, a rare parasitic condition of the tongue, the affected portion is of a brownish-black or black color. Beginning usually as a small spot it extends slowly, so that at the end of a month the dorsum is covered. The centre is blacker than the circumference. After the entire dorsum is covered the spot begins to disappear from the circumference toward the centre, and desquamation ensues. Desquamation may last from a few days to two months. The papillæ of the affected surface are enlarged and look like "a field of corn laid by the wind and rain." The sensations of taste and touch are not altered, but a feeling of dryness is marked. It must be remembered that a black tongue is sometimes the result of deliberate deception.

Movements of the Tongue.—Interference with its mobility occurs in asthenias, as in severe infections or when the mouth is dry. It may be tremulous, as in alcoholism or in simple weakness alone. It may be slow or impeded in the various stages of paralyzes. It is tremulous and the seat of fibrillar contractions in general paralysis. It cannot be protruded at all in glossolabial paralysis; it is protruded with difficulty in general paralysis, progressive muscular atrophy, and hemiplegia, because the paralysis is only partial. The tongue deviates to the paralyzed side of the body in hemiplegia with facial involvement.

Patches and Plaques.—First, there is the smokers' patch, on the middle of the dorsum about the point where the pipe-stem rests, or where the stream of smoke from the pipe or cigar strikes the tongue. This is a slightly raised area of oval shape; it is not ulcerated, but is smooth and red, or livid. Sometimes it is bluish white or pearly in appearance. The smoothness is characteristic. White and bluish-white patches or plaques, also known as opaline plaques, are seen in *leukoma*, *leukoplakia*, *ichthyosis*, and *keratosis*. These patches are unknown under twenty years of age, do not commence after sixty, and are very rare in women. They are not attended by subjective symptoms usually. There may be a sense of induration and dryness. The course is always chronic.

Eczema of the tongue—the geographical tongue—occurs most frequently in children. One or more smooth red patches are observed on the dorsum of the tongue, neither depressed nor elevated. The patch spreads and assumes a circular or oval outline; a border is faintly or decidedly yellow, usually slightly raised and sharply defined. The circles may widen and contract from time to time without symptoms. The disease may continue for months or years.

MUCOUS PATCHES.—Mucous patches are multiple lesions of syphilis in the mucous membrane. They have been referred to in the section on Diseases of the Mouth.

Ulcers of the Tongue.—*Simple* ulcers follow long-standing superficial glossitis. The ulcer is smooth, red, and glazed on the surface; the edges are callous and inactive, and the shape irregular.

Dyspeptic ulcers occur on the tip of the dorsum near the tip. The ulcers are small and superficial, without definite shape or character, except that they are red and irritable.

Aphthous ulcers are seen in children and adults, and when multiple are attended with fever. The foul odor of the breath is characteristic. Single ulcers are usually due to indigestion, or, in women, appear at the time of the menstrual flow.

Traumatic ulcers from the irritation of sharp teeth may persist a long time if the general health is bad. When indolent they may be mistaken for syphilitic or tuberculous ulcers. The rapidity of formation, the location opposite a rough tooth, and the absence of other signs of syphilis point to the correct diagnosis. *Chancre* is recognized by the induration, the circumscribed character of the lesion, its seat near the tip, and its association with enlargement of the lymphatic glands.

Tuberculous Ulcer.—The tuberculous ulcer presents an uneven, pale, flabby surface, covered with yellowish-gray, viscid, or coagulated mucus. The edges are sometimes sharp cut, sometimes bevelled, seldom elevated. There is little surrounding inflammation, and the adjacent portions of the tongue are but slightly swollen. The borders of the ulcer may be sinuous, and the shape oval or ovoid, or linear. In the neighborhood of an ulcer a number of tiny yellowish-gray points may be observed. The ulcer is painful and attended by salivation.

Tubercle bacilli are found in the scrapings. Ulcers due to *lupus* are also seen upon the tongue.

Nodes.—Nodules in the tongue are always tuberculous or syphilitic.

Cysts.—Various cysts occur in the tongue. Mucous cysts and blood cysts are the most common. *Cysticercus cellulosæ* and the *echinococcus* occur rarely. *Ranula* is a cyst underneath the tongue that causes suffering from mechanical obstruction. It is easy of recognition.

Parasitic Disease.—Thrush is the most common. Other infections of the mouth usually extend to the tongue.

Appearance of the Tongue.—For convenience, the classification of Dickinson as to the appearance of the tongue in disease may be utilized.

1. *The Stippled or Dotted Tongue.*—The tongue is moist and dotted with little white points, due to an excess of white epithelium on the papillæ. It is usually seen in persons in poor health without fever or grave constitutional disease.

2. *The Dry, Stippled Tongue.*—This is found in mildly acute disease, or in cases in which the constitutional disturbance is more marked.

3. *The Stippled and Coated Tongue.*—Patients in whom this is found are very frequently the subjects of acute constitutional affections. Fever is frequently present.

4. *The Coated Tongue.*—There is excess of white epithelium on the papillæ, and the coating is continuous. The intervals between the papillæ are more commonly filled with epithelium and accidental matters than in the preceding types. It is seen in acute febrile diseases, whether dry or moist. It is associated with a great degree of prostration and pyrexia, while the saliva is absent in the larger proportion of cases.

5. *The Strawberry Tongue.*—The tongue is coated and injected; the fungiform papillæ shine through the coat, particularly at the tip and edges. It is the tongue of scarlet fever, but may be seen in any acute febrile disorder. In scarlet fever, however, it appears by the second or third day—most marked after the second. Pyrexia is common in this class.

6. *The Plaster Tongue.*—A thick, uniform coat, edges abrupt and striking, covers the tongue. The papillæ are elongated and the intervals crowded with accumulations. It is the tongue of acute febrile disease. Fever is marked, prostration a common attendant and saliva deficient. Each successive tongue group described by Dickinson has been attended by more fever, greater exhaustion, and less saliva than the preceding group, and in each the tongue becomes more and more furred.

7. *The Furred or Shaggy Tongue.*—When the tongue is moist, the papillæ are greatly elongated and composed mostly of horny epithelium. The appearance is the same as if the tongue were dry. It is seen most commonly in old age. The dry, furred, or shaggy tongue may succeed the dotted tongue or the coated tongue in the course of advancing disease. It is the result of disease and lack of moisture,

the saliva being deficient; it indicates that there has been fever, and that possibly but little food was taken. The moist, furred tongue is not so common as the latter.

8. *The Incrusted, Dry, Brown Tongue*.—Over the surface of the tongue there is a dry, thick, felted coat, which is continuous and dips down between the papillæ. In the course of fevers it is the outcome of a preceding condition, the coated tongue, and is indicative of the infectious fevers with high temperature, but may be seen in conditions of low temperature, as cancer, phthisis, chronic nephritis, and chronic nervous diseases. There is much depression or prostration associated with it, and there is absence of saliva. If the patients with a dry, brown tongue recover, the latter retrogresses to the furred or incrustated tongue, which in turn becomes bare gradually, at first in small layers.

9. *The Red, Dry Tongue*.—This indicates a more serious condition usually than the dry and brown. It is the tongue of chronic wasting diseases. It occurs in the later stages of phthisis, and like the raw-beef tongue, is associated with dysentery and liver abscess. There may be fever. It is in a measure the tongue of chronic diarrhea. The tongue is shrunken, red, polished, and smooth. The papillæ have disappeared, and the epithelium is stripped off in patches. It may be associated with aphthæ.

10. *Red and Membranous*.—There is a membrane upon the dorsum, otherwise it is like the red, dry tongue.

11. *Cyanosis, or Venous Congestion of the Tongue*.—The tongue is of a bluish or purplish color, the surface is smooth and wet, and the papillæ are almost indistinguishable. It is not confined to organic heart disease or cyanosis. It is of quite frequent occurrence in conditions associated with albuminuria. With the venous congestion in the albuminuric cases there is always a superabundance of deep epithelium. When the surface is examined it looks as if the papillæ were fused together and overlaid by a moderate coat.

The Tongue in Relation to Diseases of the Alimentary Canal.—We associate the clean, red tongue, with or without excoriations and irritated papillæ, to gastric hyperacidity; a furred tongue, to anacidity with myasthenia. Some forms of constipation are often connected with changes in the tongue, but such connection is not constant. A dry tongue is well-known to occur in acute obstruction, and is due to deficiency of salivary secretion. In chronic obstruction, unless there is constitutional disturbance, the tongue will not change. In diarrhea all conditions of dryness, furring, and incrustation are observed. The absence of saliva, dehydration, and pyrexia help the desiccation. In diarrhea, therefore, the change in the appearance of the tongue is more marked than in any other disease.

The Tongue in Prognosis and Treatment.—The condition of the tongue is due very largely to four states—dehydration, exhaustion, pyrexia, and local conditions about the mouth. The degree of fever, the state of the nervous system, the maintenance or abeyance of secretions,

and the failure of vitality are indicated by the condition of the tongue. The return of moisture, the disappearance of fur, and the subsidence of tremor indicate that these conditions are subsiding. The persistence and increase of these signs show that the patient is worse. The tongue seldom points to solitary organs or isolated disorders, but is a gauge of the effects of disease upon the system. As to indications for treatment, the dryness, the furring, and incrustation are connected with lack of saliva. Such lessened secretion of saliva, as well as other digestive secretions, is followed by loss of appetite and impairment of digestion. Hence, liquid food and stimulants are to be used. The dry and brown tongue is of serious prognostic omen in all conditions. While it may be due to want of saliva alone, it also occurs as a part of the failure of nutrition in hectic fever, suppuration, and other conditions. It is an indication for the use of tonics, stimulants, and liquid and highly nutritious food. The weak pulse does not more surely tell of an asthenic tendency than the red, dry, and polished tongue.

THE FAUCES AND PHARYNX

Method of Examination.—The unaided eye is sufficient, the throat being illuminated as in the examination of the larynx. The mouth should be opened as wide as is consistent with comfort. The tongue is pressed out of the way by the use of a tongue depressor, preferably the cheap wooden ones which are thrown away after each examination. The patient should breathe deeply and quietly as the tongue, if rigid, relaxes upon deep inspiration. Sometimes the fauces can be examined if the tongue is protruded and held with a soft napkin between the finger and thumb by the patient.

Inspection.—In examining the fauces and pharynx, observation is made of the color of the parts, the appearance of the mucous membrane and its glands, the appearance and position of the uvula, the size of the tonsils, the character of the secretions on the pharynx, and the presence or absence of swellings and abnormal exudations.

Odor of the Breath.—In follicular tonsillitis the breath has a peculiar intense and fetid odor. There is also fetor of the breath in cancer and syphilis. This symptom may be of diagnostic significance in distinguishing cancer from tuberculosis.

Color of the Mucous Membrane.—The color of the mucous membrane is generally dark red. In the acute forms of pharyngitis it is bright red. In cases of heart disease, when there is cyanosis, the veins are congested and the surface dusky. In obstruction of the superior vena cava by tumor there is a cyanotic hue of the surface of the pharynx.

Appearance of the Surface.—The capillary vessels may pulsate in aortic regurgitation. Pulsation of the internal carotid may be seen if an aneurism or aortic regurgitation is present. Bleeding-points may be seen over the surface of the pharynx, the discharges of blood from

which may simulate pulmonary hemorrhage. The blood may be swallowed and then vomited, and hence gastric hemorrhage is simulated. It is often due to adenoid vegetations in the nasopharynx. In chronic pharyngitis the membrane is dry, the glands are prominent, and the secretion viscid. Projection forward of the posterior wall may be due to retropharyngeal abscess or caries of the vertebræ.

Eruptions.—The eruption of scarlatina or measles is also seen in the pharynx, and the papules and pustules of variola are frequently observed in that situation.

Ulceration.—**Perforation.**—Perforation of the soft palate is usually the result of syphilis.

Follicular Ulceration.—Small superficial ulcers corresponding to the follicles may be seen over the posterior wall of the pharynx. They occur in chronic catarrh, and are due to inflammation of the follicles. In addition, ulcers secondary to infectious processes are sometimes seen, as in typhoid fever. In the secondary stage of syphilis, small shallow and painless ulcers are seen on the posterior wall of the pharynx and on the palate. Mucous patches are observed at the same time, not only on the pharynx, but also in the mouth. In the tertiary stage deep ulcers, followed by scars, are seen on the posterior wall of the pharynx. Although the absence of pain renders it probable that these ulcerations are of syphilitic origin, nevertheless other evidences of the disease ought to be secured before establishing a diagnosis.

Tuberculous Ulcers.—These are irregular in shape, have a grayish floor, and are extremely painful. They are associated with ulcerations in the larynx and occur in advanced phthisis.

Cancer.—Cancer of the pharynx is rare, and is usually secondary, the disease having developed in other situations.

Excudations.—On the pharynx an exudation is usually due to diphtheria. If there is any doubt as to the nature of a membrane, cultures should always be made.

Anæsthesia.—Some of the results of inspection may be confirmed by means of the probe, and alterations in the sensibility of the pharynx may thus be detected. Sensations may be absent in the whole posterior wall of the pharynx. Loss of sensation may occur in hysteria, in bulbar and diphtheritic paralysis. On the other hand there may be an apparent hyperæsthesia, as is sometimes observed in hysteria. Inflammations increase the hyperæsthesia.

Spasm of the Pharynx.—This is a subjective symptom complained of in some cases of pharyngitis. The degree of spasm or the amount of choking sensation is largely dependent upon the nervous constitution of the individual.

The Uvula.—In health the uvula hangs midway from the palate. It varies in shape from congenital causes, and may be elongated on account of disease. This takes place particularly in chronic nasal catarrh if there has been snoring or coughing. The uvula may be swollen and edematous in association with subcutaneous edema in

acute nephritis, and also idiopathically. It may become so enlarged as to interfere with swallowing and breathing. In addition to the constant cough which it causes there may be dyspnea and repeated attacks of choking. Hemorrhagic infarcts may also take place in the uvula.

The Tonsils.—The entire surface of the tonsils can be seen by ordinary inspection. If enlarged, the posterior surface cannot be seen, although a better view may be obtained by causing the patient to say "ah," during which they are brought forward to the light. They are pathologically of much importance. The crypts of the follicles open on the surface, and in disease are visible. The tissues and gland follicles are liable to inflammations which may result in the entrance of pathogenic bacteria into the system.

Acute Enlargement.—The tonsils become enlarged; the swelling takes place rapidly in the acute form. They may be simply enlarged and the covering membrane intensely red. In other forms of inflammation the surface may be dotted over with white points, due to exudation from the follicle, and may be covered with a white or grayish membrane.

Chronic Enlargement.—The tonsils are enlarged when there is general hyperplasia of the lymphatic structures. Repeated attacks of inflammation cause chronic enlargement of the tonsils. They are enlarged sometimes to a great degree, filling almost entirely the lumen of the fauces. The surface is irregular and may be scarred. The mouth of the follicles may be dilated. Mouth-breathing is present. The tonsils may be the seat of *sarcoma* and *tuberculosis*.

Ulcers.—Tuberculous ulceration is rare. Deep ulcers on both surfaces are usually syphilitic, though a superficial ulceration occurs in those rare cases of tonsillar chancre. An irregular spreading ulcer with fetor and sanious discharge is usually cancerous. The lymph nodes are enlarged.

Exudations.—Exudations on the tonsils are due to diphtheria, to a streptococcic infection, to the inflammation which attends scarlatina or which arises secondarily to other infectious diseases, and to fungi.

In healthy persons the foul-smelling plugs which block the tonsillar crypts are found to be made up of pus cells, broken-down epithelium, bacteria, and *Leptothrix buccalis*.

THE ESOPHAGUS

Stiffness of the neck is seen in acute inflammation of the esophagus in periesophageal abscess, and in traumatism.

The *expectoration* in diseases of the esophagus is characteristic. It is usually a glairy mucus, often frothy or viscid. It is not coughed up, but after welling into the pharynx is hawked up. It is abundant in acute and chronic inflammation and in cancer.

Hemorrhage from the Esophagus.—Hemorrhage from the esophagus occurs from varicosity of the veins at the lower portion of the gullet.

It may occur in old people, or at any age in cirrhosis of the liver. In hemorrhage from the esophagus the blood is usually bright in color, has not been acted on by an acid, as in hematemesis, and is, therefore, alkaline in reaction, and is not discharged by vomiting, although vomiting may occur after the blood is poured out. In a grave case of purpura under the care of the writer, hemorrhage took place from the lower end of the esophagus. Small bleedings from the esophagus are usually indicative of cancer, especially if, in addition to the hemorrhage, there are present the symptoms of occlusion. Hemorrhage may also be due to foreign bodies, trauma, and ulceration.

Emaciation is the most characteristic general symptom of esophageal disease. It is, of course, more striking in cancer, but occurs to a moderate degree in all forms of stricture.

Fetor of the breath attends dilatation of the esophagus.

Emphysema of the subcutaneous connective tissue should always lead to investigation of the esophagus. Usually it is found to have been preceded by pronounced symptoms of disease of the esophagus. In rare cases ulceration of the esophagus may progress without symptoms, and extend into the air passages. The passage of air through the fistulous communication causes subcutaneous emphysema. It is of frequent occurrence when foreign bodies lodge in the gullet.

Physical Examination.—Examination of the esophagus is made by inspection and auscultation, and by means of palpation with or without a bougie. Inspection can be made with the esophagoscope, for with this instrument it is possible to study the condition of the mucous membrane, to locate strictures and dilatations, to excise pieces of ulcerated tissue for microscopic examination, and to locate and frequently remove foreign bodies.

Auscultation.—Auscultation of the esophagus, while the patient is swallowing fluids, normally reveals two sounds. One occurs after the patient swallows, and has no clinical significance. The second is heard normally after an interval of about six seconds, and is due to the contraction of the esophageal muscle forcing the fluid onward through the cardia. This sound is delayed or entirely absent in stenosis of the cardia or when obstruction along the course of the esophagus has caused great relaxation of the wall above. The proper points for auscultation are to the left of the ninth or tenth dorsal vertebra, or to the left of the tip of the ensiform cartilage.

Palpation.—The esophagus behind the trachea in the neck may be palpated when it is enlarged, as in abscess and in cases of diverticulum of the upper portion of the tube. It may be possible by pressure to empty the contents of a diverticulum into the esophagus, and thus to cause the disappearance of the palpable mass.

Percussion.—Percussion of the neck, or along the spine, in cases of sacculated diverticulum, shows a localized dullness, which is often absent when the sac is empty.

X-ray.—The Röntgen rays may be used with success to demonstrate the presence of dilatation above the stricture, and, more especially, sacculated diverticula in the lower portion. When empty, the sac gives a bright area; when filled with bismuth or other metallic solutions, it gives a deep shadow of regular outline.

Sounding.—The normal constriction of the esophagus is situated nearly opposite the fourth dorsal vertebra, ten inches from the teeth. If dysphagia is due to paralysis or spasm of the esophagus, the bougie can usually be passed with ease. If, on the other hand, it is due to organic disease, an obstruction will be found, which is generally in the upper half of the esophagus. If near the pharynx, the obstruction is due to cicatricial stricture. If the obstruction is encountered nine inches from the teeth or about the position of the bronchus, it is usually due to cancer. The bougie should not under any circumstances be passed if there are grounds for believing there is an aneurism. Fatal rupture has followed its passage under such circumstances.

Method of Examination.—The patient should be seated with the head thrown back sufficiently far to make the passage from the pharynx to the esophagus almost continuous. The operator may stand behind or in front of the patient. The bougie, held like a pen, should be passed through the pharynx, guided by the fingers, close to its posterior wall. But little force should be used. It should be passed slowly, for then the gagging will soon be overcome. The bougie should be warmed and oiled before it is introduced. The handle should be flexible whalebone, and the bulb olive-shaped. The stomach-tube is a sufficiently accurate sound for ordinary purposes.

CHAPTER XVII

EXAMINATION OF THE NERVOUS SYSTEM

General Consideration.—The nervous system is composed of two parts, the cerebrospinal and sympathetic. Through these parts every function and act of the body, whether voluntary or involuntary, conscious or unconscious, is directly controlled so that the integrity of the central nervous system is necessary in order to perform properly the various functions and actions of life. The cerebrospinal system innervates the skeletal muscles. The sympathetic portion conveys impulses to and from the thoracic and abdominal organs; the involuntary muscles and glandular tissue throughout the body and the heart muscle. Though the two systems are intimately associated very little is known of the pathology of the disorders of the sympathetic system, and still less of their clinical manifestations, while the recognition of lesions in the cerebrospinal system has reached a high degree of accuracy.

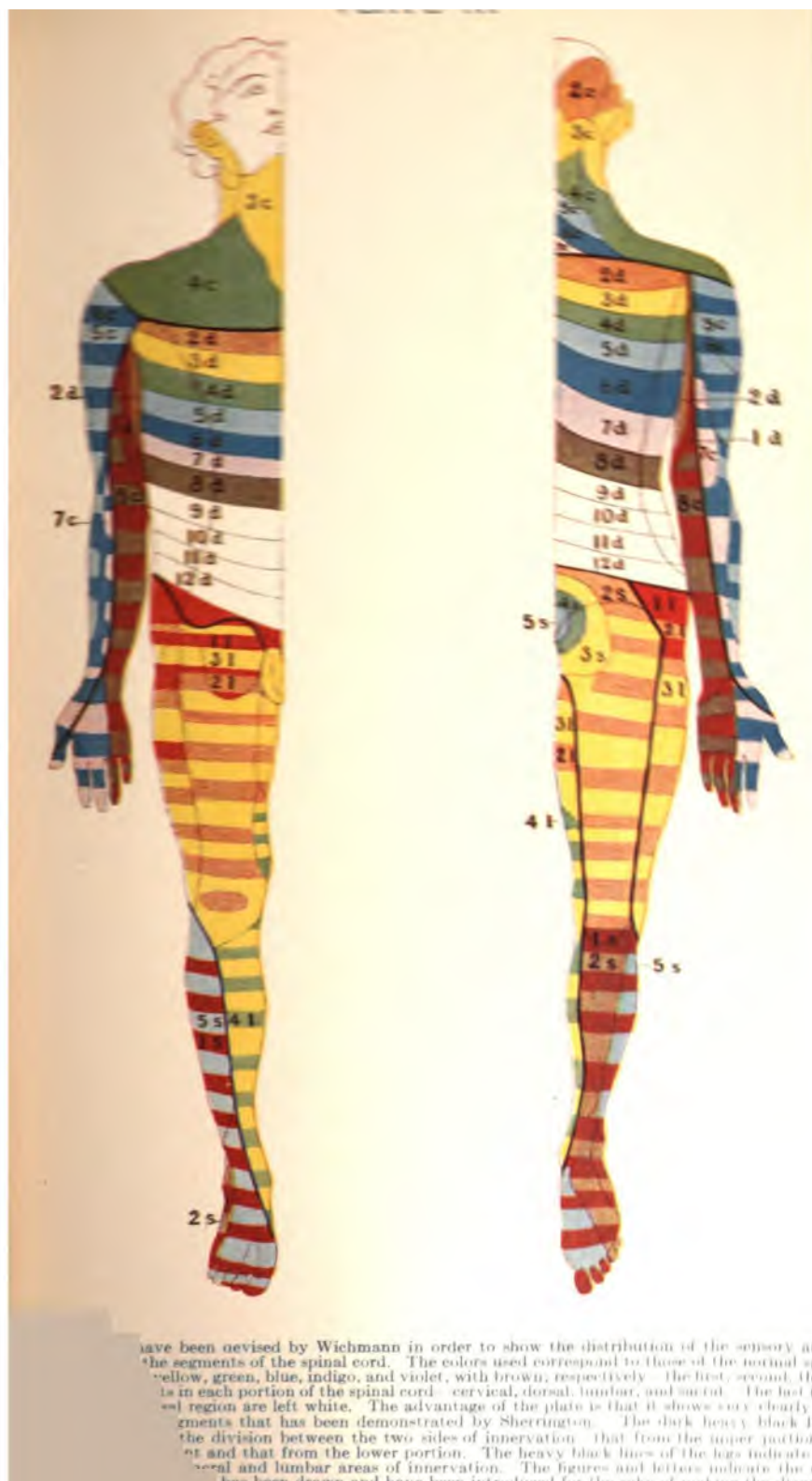
The nervous system may be regarded physiologically as a collection of neurons. By *neuron* is meant a nerve cell and all its processes to their ultimate ramifications. The processes are of two kinds: the so-called dendrons which are relatively short, thick, and branched, and appear to resemble in many respects the protoplasm of the nerve cell itself; and the axon, or axis-cylinder, a long slender process that in its course gives off at intervals still more slender branches, the collaterals, which communicate with the collaterals of other neurons, and at its termination usually break up into a small tuft of fibers, the terminal arborization, which connect either by continuity or contiguity with the tendons of some other ganglion cell, or end in the peripheral muscle fibers. At a certain distance from the nerve cell the axon usually becomes surrounded by myelin sheaths, and constitutes the nerve fibers which make up the greater bulk of the central nervous system (the white substance) and practically all of the peripheral nervous system. Neurons with similar functions are usually grouped together, the aggregation of the cells forming a nucleus, and of the fibers a bundle, tract, or system. The gray matter is largely composed of these groups of ganglion cells or nuclei. The axons convey impulses from the cell, and the dendrons convey impulses or nutriment to the cell. In the cell itself these impulses are modified or altered in some as yet unknown manner. The functions of the neurons are sensory, motor, trophic, and secretory. Those which have been most accurately studied may be divided into the sensory neurons, conveying impulses from the peripheral nervous system and the motor neurons, conveying impulses from the central nervous system to the muscles.

Sensory Neurons.—Impressions of touch, muscle sense, pain, heat, and cold are conveyed by the sensory nerves or neurons (dendrons) from the skin, muscles, mucous membranes, tendons, and joints of all the body except the parts supplied by the cranial nerves. The sensory neurons commence as tactile corpuscles. From them the dendrons pass through the peripheral nervous system to the posterior roots of the spinal cord, and here each enters a cell in the ganglia of the posterior roots. From these cells a fiber emerges that for a short distance is continuous with the entering fiber, and then leaves it and continues along the posterior root of the spinal cord, here it divides into two branches, an ascending and a descending branch. Of the function of the latter nothing certain is known. Some of the ascending branches pass into the lateral posterior column (Burdach) and at a higher level into the median posterior column (Goll). Those entering the cord in the upper dorsal and cervical regions, however, continue in the lateral posterior column. Both columns end in the nucleus cuneatus and the nucleus gracilis respectively. These two groups of fibers probably only convey tactile and muscular sensations. The fibers conveying pain and temperature sensations apparently pass up the cord through the central gray matter, but their central terminations are not yet definitely known. From the ganglion cells in the two nuclei in the medulla, axis-cylinders arise that pass toward the brain and form a mass of fibers known as the fillet. They occupy the central regions of the pons in its posterior part, but anteriorly they spread gradually out until they form a narrow band, placed horizontally, just below the gray matter surrounding the aqueduct of Sylvius. They then enter the tegmentum of the crus, and the majority lose themselves in the ventral nucleus of the optic thalamus. They constitute the second chain of sensory neurons. It is probable that from the optic thalamus, and from the other nuclei in which perhaps fibers of the fillet terminate, other axis-cylinders arise which pass through the corona radiata to the sensory areas in the cortex. These sensory areas will be discussed in connection with the cortical localization.

Destructive lesions in the peripheral sensory nerves produce total anesthesia of the part supplied. Partial lesions may produce partial anesthesia or even dissociation of sensation. Irritative lesions of the peripheral nerves produce severe pain, usually referred to the part supplied by the nerve, and there are also sensitive points or general tenderness over the nerve trunk. Certain forms of irritative lesion produce partial alteration of sensation, which is usually spoken of as paresthesia (*q. v.*). Trophic changes in the skin often occur. Destructive lesions of the posterior roots also produce total anesthesia. If the lesion is on the peripheral side of the ganglion, there are in addition trophic changes in the part supplied. Lesion of the ganglion itself usually produces anesthesia and trophic changes, if complete; if partial, the symptoms are variable. In some cases herpes zoster along the course of the nerve has been observed. Irritative lesions of the posterior

roots produce fulgurant pains in the limbs, or a feeling of constriction in the trunk. They may also be the cause of visceral crises. Destructive lesions of the posterior columns of the spinal cord produce more or less tactile anesthesia and loss of the muscle sense. As a result of the latter there is ataxia. Lesions of either of the two central sensory neurons produce various forms of anesthesia, depending upon their extent. According to our knowledge of this subject, destructive lesions, such as hemorrhage in the posterior portion of the posterior limb of the internal capsule, or destructive lesions of the optic thalamus are usually associated with hemianesthesia on the opposite side of the body. At times tactile sense is preserved and only the pain sense is lost; as a rule, however, all forms of sensation are more or less affected.

Motor Neurons.—The motor neurons consist of two groups, the central or upper, and peripheral or lower neurons. The central motor neurons commence in the motor portion of the cortex. They then pass through the corona radiata to the internal capsule, where they form a large band of fibers occupying the knee and the anterior two-thirds of the posterior limb. The fibers for the face occupy the knee and anterior third of this portion. Next come the fibers for the arm, then those for the leg, and, finally, the fibers for the trunk. From the internal capsule the fibers pass into the crura cerebri, where they lie beneath the substantia nigra, occupying about the middle of each crus. The fibers for the face and cranial nerves lie internal to those for the extremities and trunk. From here they pass to the ventral portion of the pons, where they are broken up into small bundles by the association of fibers of the two cerebellar hemispheres. These reunite and form the pyramids in the anterior portion of the medulla, which decussate in the first cervical segment and pass down the cord as the lateral pyramidal columns. A few of the other fibers, however, do not decussate at this time, but pass downward in the direct pyramidal columns which decussate through the anterior commissure of the cord at lower levels. The fibers for the cranial nerves decussate, as a rule, in the neighborhood of the nuclei for these nerves. The fibers for the oculomotor nerves decussate in the tegmentum and the nuclei around the aqueduct of Sylvius. The fibers for the facial decussate in the anterior portion of the pons. From this point downward fibers are continually crossing the median raphe to the nuclei of the various motor cranial nerves until the median decussation—that is, in the first cervical segment. It follows, therefore, that if a lesion occurs in such a position that it affects the fibers of one of the cranial nerves after they have crossed the median line, at the same time involving the undecussated fibers of the pyramids, we will have the syndrome known as crossed paralysis—that is, the muscles supplied by the affected cranial nerves will be paralyzed on the same side as the lesion, and the rest of the body on the opposite side. (See Lesions of the Cranial Nerves.) The peripheral motor neurons commence in the cells of the anterior cornua



of the spinal cord, passing out through the anterior roots, and reach the muscles through the peripheral nerves.

The functions of these two sets of neurons are not identical. The central motor neurons convey impulses from the cortex to the cells of the anterior cornua, by which the latter are stimulated to produce muscular movement. At the same time they seem to possess an inhibitory influence by means of some constant activity, so that while they are intact the reflexes are restrained and the muscles do not become spastic. Upon the nutrition of the muscles they apparently have no influence whatever, or at least act only indirectly by causing paralysis. The peripheral motor neurons control directly muscular activity. By their continuous action they maintain muscle tonus, and when unrestrained by the influence of the upper neurons, produce a condition of spasticity. While they and the sensory neurons forming the arc are intact, reflex action persists. They also control in some unknown way the nutritional changes in the muscles. Destructive lesions of the lower neurons cause paralysis and degenerative change in the muscles. Irritative lesions cause spasms, usually tonic in character, and either momentary (as in facial tic) or persistent (tetanic). The muscle tonus is lost, and the paralysis is, therefore, flaccid in character, while the reflexes are abolished. Destructive lesions in the central motor neurons, on the other hand, produce paralysis of the muscles; but their nutrition is not impaired, their muscle tonus is increased to spasticity, and the reflexes are exaggerated. Irritative lesions of the central motor neurons produce, as a rule, clonic spasms. These may be limited to the part irritated, as occurs in some forms of central softening in the motor region or become generalized. (See Convulsions.)

Disturbance of Sensation.—In testing any of the forms of sensation, certain general methods should be employed. It is usually best to approach a suspected anesthetic area from an area where sensation is normal. The boundary marks should be made with any suitable utensil, and then this point tested again by passing from the anesthetic to the normal area. As a rule, the transition is so distinct subjectively that there will be no difficulty in outlining the area by approaching it from various directions and by passing from its centre in the reverse manner. As soon as the examination is complete, the results should be recorded upon a diagram. This is really the only way in which they can subsequently be accurately studied, and often a rough outline sketch is more satisfactory than any amount of verbal descriptions. When the results have been obtained, it must first be determined whether their distribution corresponds to the distribution of the cutaneous nerves or to the sensory areas supplied by the segments of the spinal cord. This is usually easy, and is greatly facilitated by reference to Plate III. It must then be determined what nerves or segments are involved and a careful comparison made between the sensory and motor changes in order to determine either agreement or divergence.

If they agree, the diagnosis is, of course, readily made; otherwise it is sometimes difficult to determine exactly what nerve or segments have been affected. (See section on Localization of Lesions of the Cord.)

Simple Sensations.—Tactile Sense, Esthesia, or the Sense of Touch.—This is the ability to know when some external object has come in contact with the skin. It may be tested in a variety of ways. For ordinary clinical work it is sufficient to touch the skin lightly with the tip of the finger. A blunt instrument may also be employed, or, if sensation is still acute, a camel's-hair brush or cotton point. If a hard instrument is employed, the investigator should be careful that it is not sharp or rough, so that the pain sense may be excluded, and also that no force is used in applying it to the skin in order that the pressure sense may not be involved. The patient should close his eyes, or, what is better, permit them to be bandaged, and should then be instructed to indicate by some word or gesture the moment contact takes place.

Hyperesthesia.—This is an increased sensitiveness to contact. Its most common cause is functional exaltation or irritability of the nerves as in neuralgia or neuritis. Occasionally functional conditions, such as neurasthenia and hysteria, may be merely the result of some local irritation of the skin. It also occurs in organic diseases of the cord, and is then limited to the area of distribution of the spinal segment just above the destructive lesion—the zone of hyperesthesia.

Hypesthesia.—This is a decreased sensitiveness and occurs in neuralgia, in partial lesions of the spinal cord, particularly disease of the posterior columns, and rarely in cerebral lesions of various kinds, particularly those occurring in the parietal lobe, in the end of the posterior limb of the internal capsule, and in the pons. It is also found in functional nervous conditions.

Anesthesia.—This is a total loss of sensation and results from solutions of continuity of the sensory nerves, from destructive lesions of the cord or from brain lesions. It may be functional, involving irregular areas, and as such is the commonest form of hysterical stigma. Organic anesthesia may be distinguished from functional anesthesia by its distribution, an area of anesthesia in segments, that is, bounded by two horizontal lines passing around the body, is due to disease of the cord. Anesthesia limited to the opposite of the body occurs in unilateral lesions of the cord. In cerebral diseases anesthesia is commonly unilateral and corresponds to the paralyzed side or extremity, if paralysis is present.

Trichesthesia.—Simple sensation of perhaps less clinical importance than the foregoing is trichesthesia, or the consciousness that a cutaneous hair has been touched. Von Bechterew calls attention to the fact that trichesthesia and tactile sense are not equally delicate in various parts of the body.

The Ticking Sense.—This is not clearly understood. It may be described as an involuntary effort to escape rapidly repeated slight

stimuli. It is diminished in conditions that diminish tactile sensation, but otherwise has no clinical significance.

Pain Sense or Algesia.—This is the ability to perceive pain of any kind. It may be produced by various kinds of irritants, such as cutting, bruising, caustics, electricity, etc., to each of which the response varies. For clinical purposes it is sufficient to use a needle or pinch a small fold of skin between the finger nails.

Unlike touch, pain can also be elicited by irritation of the nerve fibers that convey it to the cord, or perhaps of other nerve fibers that exist in the tissues and in the nerves themselves. The sensation produced by this form of stimulation is somewhat different from that perceived when the pain terminals alone are irritated, and is either of a rending or boring character. It can be elicited most readily by pressure upon a nerve trunk, particularly when it crosses a bone, as, for example, the supra-orbital or the ulnar. The paresthesias may, in cases of special intensity, be extremely painful.

Visceral Pain.—This may be elicited by strong pressure upon the testicles, ovaries, or breasts, or by a violent blow upon the abdomen. It is usually characterized by intense prostration and nausea. Visceral analgesia occurs in some cases of *tabes dorsalis* and occasionally in hysteria.

Hyperalgesia.—An increased susceptibility to painful impressions, so that the lightest contact may cause exquisite agony, occurs in inflammation, and in those conditions associated with hyperesthesia. A variety of hyperalgesia is *tenderness*—that is, pain elicited by simple pressure. It is most frequently associated with local inflammations, and occurs along the course of the nerves in neuritis and neuralgia.

Hypalgesia.—This is decreased susceptibility to pain and occurs as a result of partial lesion of the nerves or of the central portion of the spinal cord, and occasionally as a result of focal lesions in the brain. It is also very common among idiots, immediately after epileptic attacks, and in cases of hysteria.

Analgesia.—Insusceptibility to pain is an exceedingly important symptom. Analgesia of organic origin results from local destruction of the nerve, from disease of the central gray matter of the spinal cord, such as occurs in transverse myelitis, in syringomyelia, and in tumors of the cord, and from focal disease of the brain, particularly if situated in the parietal lobe, or in the posterior limb of the internal capsule. It also occurs in a great variety of functional conditions, and may be general in certain forms of insanity. Organic analgesia is frequently associated with trophic changes, either as a result of the inability of the part to defend itself against irritation, or as a result of the intimate association of the sensory and trophic nerve fibers.

The Sense of Roughness.—This is allied to the pain sense: it serves to estimate the degree of friction between the skin and an external body. It appears to be diminished in *tabes* and peripheral neuritis, but otherwise it is of no clinical value.

Temperature Sense, or Thermoesthesia.—This enables us to recognize the difference in temperature between various bodies. *Method:* It is usually tested by filling two test-tubes, one with hot and one with cold water, and applying them in irregular alternation to the region under investigation. In health a difference of 1° C. can be recognized upon the more sensitive portions of the body (the anterior surface of the forearms, the skin of the face, and the chest). A rough test is the use of metal and wooden objects. The *heat sense* is more complicated, and is not yet thoroughly understood. There seems to be special points upon the skin where the nerves of heat and cold terminate (Goldscheider). There may be loss of perception for cold objects, while perception for hot objects remains unimpaired, or the reverse may be present. Sometimes the patient calls all objects warm, and at other times he calls them cold.

Hyperthermoesthesia and Hypothermoesthesia.—These are practically of no value as clinical signs, for our methods of testing the delicacy of the sense are at present imperfect.

Thermoanesthesia.—Complete loss of the heat sense is very important clinically. It occurs in neuritis or destructive lesions of the nerves, and in central disease of the spinal cord, such as transverse or pressure myelitis, tumor, and especially in syringomyelia. As a result of being most frequently associated with spinal cord disease, the thermoanesthetic area is usually segmented. The heat sense may, in connection with other forms of sensation, be diminished in functional nervous diseases.

Paresthesias.—Subjective sensations, such as numbness, tickling, itching, formication, etc., which do not correspond to any external stimulus, may or may not be due to lesions of the nervous system, depending on whether they are located in a certain nerve or definite portion of the body.

Sensation of Locality.—When any part of the surface of the body is touched, we can, under normal conditions, tell the location of the point of contact. This varies considerably, however, in various parts of the body, being more accurate on the lips and less on the skin of the back between the shoulder-blades, where an error of from 6 to 7 centimeters is within normal limits. It may be very much disturbed without loss of the delicacy of the touch sense. It may be tested by making contact with the finger or any blunt object, and directing the patient to close the eyes and indicate the point touched. The sensation of locality may be diminished in all forms of hypesthesia, especially that associated with central disease, and without hypesthesia in disease of the cord, such as tabes, and in injuries to the parietal lobe. In organic disease of the cord the error in localization may be very great, and in some cases amounts to a false localization, thus irritation of the hand will be felt in the shoulder, or of the foot in the thigh. When a single contact is perceived in several places, the term *polyesthesia* is employed.

Allochiria is a term employed to describe the reference of a sensory stimulus to the corresponding location on the opposite side of the body. It may be a stigma of hysteria or is associated with some organic lesion of the cord, such as tabes, myelitis, etc.

Pressure Sense.—The clinical significance of this has not yet been determined. It may be tested by using a series of little blocks that can be piled one on top of another, or by filling a vessel more or less completely with shot or mercury, and placing them on a part so placed that it is impossible to make muscular effort.

Sense of Muscular Resistance.—This may be tested by means of weights or springs. Under these circumstances the limb should not be supported, but the patient should be instructed to hold the different weights in the hand to estimate as nearly as possible their relative value. It seems to be wholly or partly lost in cases of loss of muscle sense, or in cases in which the muscular sense is extremely impaired, as in ataxia, monoplegia, etc. The *sexual sense*, and the sense of pressure upon the sphincters—that is, the desire to micturate or defecate—is diminished or lost in various disorders of the spinal cord.

Sensation of Vibration.—This is tested by means of the vibrations of a tuning-fork placed over superficial bones. Bony anesthesia occurs in tabes, syringomyelia, and myelitis, while bony hyperesthesia occurs in tabes.

Dissociation of Sensation.—This occurs in neuritis, but is exceedingly rare. It also occurs in various forms of myelitis, particularly pressure myelitis, and is the most characteristic symptom, and for a long time was considered pathognomonic of syringomyelia. In this form of dissociation tactile sense is preserved, and the temperature and pain senses are lost. But it is now known that this type occurs whenever the gray matter of the cord is exclusively involved. In cases of partial but extensive lesions of the cord it may coexist with complete anesthesia in neighboring areas. When the tactile sense is lost, and the pain sense still present, the condition is termed *anesthesia dolorosa*. It usually occurs as a result of partial injury to a peripheral nerve.

Complex Sensations.—These are probably very numerous, but only two have been so carefully studied that they are available for clinical purposes. These are the so-called position or muscular sense and the stereognostic sense.

The Position or Muscular Sense.—By this we mean the ability to perceive and recognize the position of the limbs or of the body. It probably depends upon the coördination of the complex perceptions received from the muscles, joints, periosteum, tendons, and skin. It may be tested by having the patient perform certain movements with closed eyes or to locate the positions of the limbs after passive movement. This sense is lost when for any reason there is total anesthesia of the part, and may disappear as an isolated symptom in case of disease of the posterior columns, or in the ataxia due to central lesions.

Stereognostic Sense.—By this we mean the ability to recognize the shape, consistency, surface, and nature of any object placed in the hand or brought in contact with the skin of any part of the body. This sense depends upon a variety of perceptions. The stereognostic sense is always abolished when the tactile sense is absent. Occasionally in hysteria the patient may declare himself unable to perceive touch when the stereognostic sense is intact, but this is an exception. It may, however, be lost when tactile sense is still preserved, especially if the locality sense and the muscle sense have been greatly impaired. When due to organic causes, its absence usually indicates a lesion in the parietal lobe or in the projection fibers coming from that region. It occurs frequently in hemiplegia, in cerebral monoplegia, and occasionally in peripheral palsy, involving two forms of sensation. It has also been observed as a transient symptom after brain shock without disturbance of any other sense.

Delayed Sensation.—The interval of perception of sensation may, in extreme cases, be several seconds. It is not known where this delay takes place, whether in the sensory bodies of the skin, or in the nerves, or in the central nervous system. This symptom is manifested particularly in tabes dorsalis, but may occur in functional nervous diseases and in various forms of organic central disease. It has also been noted in peripheral neuritis. The delay can occur for one sensation alone, as the pain sense, even when tactile sense is normal.

After-sensation.—A prolonged sense of pain as following a pin-prick, is seen in disease of the spinal cord, particularly tabes, or of the nerves.

Disturbances of Motility.—These may be grouped under three heads: (I) Motility decreased, either partially, paresis; or completely, paralysis. (II) Motility incoördinated. (III) Motility increased.

I. Motility Decreased.—**PARALYSIS.**—This is a complete loss of voluntary motor power. Paralysis is called monoplegia when one extremity is involved; hemiplegia, when half of the body is involved; paraplegia, when two symmetrical extremities are involved (this term is usually restricted clinically to paralysis of both legs); paraplegia cruralis if the legs are affected; paraplegia brachialis if the arms are affected; diplegia, double hemiplegia, when two extremities are affected without involvement of the trunk. Crossed paralysis is a term applied to paralysis of one side of the face and the opposite side of the body. Local paralysis, or palsy, is the term used when only small groups of single muscles are affected, and is produced by lesions of the peripheral neurons. Paralysis is also called, according to the seat of the lesion, cerebral, pontine, bulbar, spinal, neural, muscular, or myopathic paralysis.

Varieties of Paralysis.—Paralysis may be either spastic or flaccid. In the former there is distinct resistance to passive movements, in the latter the part is limp, flaccid, and toneless, and offers no resistance to passive motion. *Spastic (central, cerebral, tonic) paralysis* is characterized by exaggerations of reflexes, increased muscle tone, absence of

the reactions of degeneration, very gradual if any muscular atrophy, and by a tendency to the development of contractures. It is usually due to some lesion in the upper motor neuron. The lesion may be situated in the cortex, the corona radiata, the internal capsule, the pons, the pyramids of the medulla, or in the lateral columns of the cord.

Flaccid (peripheral, spinal, atrophic) paralysis is characterized by absence of reflexes, diminished muscle tone, the presence of reactions of degeneration, rapid wasting, and absence of contracture. It is commonly due to lesions of the peripheral motor neurons—that is, from the anterior cornua of the cord to the muscle itself. It may, therefore, be produced by destruction of the ganglion cells, by injury to the anterior roots or to the peripheral nerves, or by disease of the muscle. Flaccid paralysis frequently occurs as the result of functional conditions—for example, it is the type of paralysis that is usually observed in hysteria.

Diagnostic Significance of Paralysis.—*Monoplegia* may be caused by small lesions in the cerebral cortex or the corona radiata. It is rarely produced by lesions of the internal capsule or of the spinal cord. It occurs in circumscribed forms of infantile paralysis, and in lesions of the peripheral nerves, particularly the roots or the plexuses. Monoplegia also occurs in hysteria and in the pseudoparalysis due to localized disease of the muscles or joints.

Hemiplegia.—This is commonly cerebral and due to a lesion of the opposite side of the central convolutions. This lesion may either be extensive and destroy the motor portion of the cortex or corona radiata, or more circumscribed, involving the internal capsule, the crura, the pons, or the medulla. In hemiplegia if the muscles of the trunk and those supplied by the upper branch of the facial nerve escape and the lower half of the face and the arm and leg are paralyzed, the lesion is one of the upper motor neurons of the opposite side, and hence cerebral. If the arm and leg on the side of the lesion and the lower half of the face on the opposite side are paralyzed (crossed paralysis, pontine palsy), the lesion is of the pons opposite the paralyzed arm and leg but below the decussation of the facial fibers, that is, in the posterior half. Lesions of the medulla, in addition to the motor tracts, involve other important nuclei and tracts.

Spinal Hemiplegia.—This is characterized by the absence of facial involvement and sensation is lost on the opposite side. It is due to a unilateral lesion above the fourth cervical segment. It is rare.

Hysterical Hemiplegia.—This can only be recognized in some cases by the discovery of the other stigma of that disease. It is almost invariably flaccid, and contractures rarely appear.

Paraplegia Cruralis.—This is usually produced by a lesion of the spinal cord. It, therefore, occurs in transverse or pressure myelitis, in tumor of the spinal cord, in hemorrhage into the spinal cord, and as a result of traumatism. It is occasionally produced by multiple neuritis of the legs, particularly that form known as Landry's paralysis, or in

Localized paralysis by lateral cerebral lesions, and occasionally as a functional condition. The type of paralysis is spastic if the lesion is situated above the lumbar portion of the cord. The type is flaccid and the reflexes are abolished if the lesion is in the lumbar or sacral region, or involves the cauda equina.

Paraplegia Brachio-cephalica.—This is a rare condition, occurring chiefly as the result of a localized meningitis in the cervical enlargement, particularly poliomyelitis hypertrophica cervicis. It may also occur in syringomyelia and more rarely as a result of traumatic injury to both sides of the cervical plexus ("crutch palsy.")

Paraplegia Caudalis.—This is almost invariably the result of bilateral spinal injury which may occur as a result either of neuritis or of an injury to both sacral nerves after they leave the medulla.

Local Paralysis.—These are usually due also to lesions of the peripheral nervous system. They are commonly the result of some trauma injuring a single nerve trunk. The paralysis is flaccid, and the reactions of degeneration are present.

Multifocal Paralysis.—They are usually due to some general condition affecting the peripheral nervous system. The paralysis is usually flaccid and incomplete, that is, certain groups of muscles escape. In polyneuritis due to intoxication or infection there may be paralysis either of certain groups of muscles, particularly the extensors, or of the entire limb. This occurs most frequently in poisoning by lead, arsenic, and alcohol, in chronic diseases, as diphtheria or in beriberi. The paralysis is usually always flaccid, there is muscular atrophy, and the reactions of degeneration ultimately appear.

Myopathic Paralysis.—Congenital absence or complete atrophy of the muscle gives rise to myopathic paralysis.

Paresis.—This is a term used to indicate partial loss of voluntary motor power. In addition to the causes given for paralysis, paresis may be produced by exhaustion or by profound emotion. There are two forms of paresis—one in which the muscle is unable to exert its normal force at any time, and the other in which the muscle may exert its normal force for a brief period, and then rapidly become exhausted and insufficient. In the former there is some deformity, such as foot-drop or wrist-drop. In the latter the symptoms do not appear until some effort has been made. Paresis may also be temporary, as after emotion or fatigue; stationary, as in cases of injury to the central nervous system; or progressive, as in the myopathies. In the latter condition the muscles waste and lose their power, but reactions of regeneration do not occur, and there are no fibrillary twitchings. Ultimately, the condition may go on to absolute paralysis. The power of the muscles may be tested very accurately by means of the dynamometer, or by the power of resistance of the patient to passive movements, such as extension of a flexed arm.

II. Motility Incoördinated.—**ATAXIA**.—This means the inability to coördinate perfectly. Perfect coördination depends upon many factors,

such as the muscle sense, visual impulses, and vestibular control, but the practical testing for ataxia takes place in the examination of the motility. Various types of ataxia have been distinguished: *spinal ataxia*, in which the disturbances of motion are more pronounced when the eyes are closed, and which is due to disease of the posterior columns; *cerebellar ataxia*, in which the disturbances are equally severe when the eyes are opened or closed, but which disappear when the patient lies down; *cerebral ataxia* which occurs as a result of injury to the parietal lobe, and is accompanied by loss of muscular sense and marked persistent incoördination of movement; *pseudo-ataxia*, due to weakness of certain groups of muscles, so that they do not properly oppose the action of other groups. Finally, there is a form of ataxia apparently due to anesthesia of the skin and loss of the muscular sense, in which the patient is able to perform movements perfectly as long as he can watch the part with the eye, but as soon as the eyes are closed the ataxia appears.

Ataxia may be tested in a variety of ways. Ataxia of the upper extremities may be recognized by directing the patient to touch the tip of the nose with the tip of the forefinger. The ataxia of the legs may be tested by requesting the patient to bring the heel of one foot against the knee of the other.

Romberg's Symptom.—If the ataxia is very slight, it may be necessary to have the patient stand with the eyes closed and feet together, or to attempt to step backward under the same conditions. Under these circumstances the swaying is more pronounced than that noticed when a normal person attempts to perform the same movements. If the ataxia is at all severe, it produces a characteristic disturbance in the gait. (See Ataxic Gait.)

III. Motility Increased (Spasm).—Spasm is a condition in which a muscle or group of muscles are involuntarily contracted: (A) either persistently (tonic or tetanic spasm) or (B) rhythmically (clonic spasm).

The term convulsion is used to designate general spasm with loss of consciousness. It is often incorrectly employed to indicate general clonic spasm of the whole body, even when consciousness is still present. General convulsions invariably indicate some disturbance in the brain; whether it be some chronic disease with occasional exacerbation of cortical irritation, some acute injury, or some systematic condition, such as diabetes or uremia, or a severe infection. A functional disturbance may be hysteria or epilepsy.

A. TONIC SPASMS.—These are characterized by vigorous contraction of the muscle, which becomes hard and painful. They are due to overstimulation of some portions of the motor tract in the brain or cord. Tonic spasms can usually be diagnosticated by simple inspection. They occur particularly in tetanus, meningitis, strychnine-poisoning, and hysteria. Localized spasms in the upper extremities may occur as a result of disease of the cord above the cervical enlargement or of the brain, producing a spastic condition of the muscles, which, however,

acterized by clonic spasms of a group of muscles which may be alone affected or the spasms may affect consecutively the muscle groups that are controlled by centres adjacent to the primary centre affected. Thus if the facial centre is first involved, the face, then the hand and arm and later the leg on the same side are thrown into convulsions. The spasms are usually limited to one side of the body and the patient rarely loses consciousness.

Tremor.—This is a disturbance of motion characterized by an oscillation of the part or parts involved. Tremor may be of various kinds. It may be fine or coarse, constant or irregular. It may disappear upon voluntary effort, or only be apparent when motion is attempted (intention tremor). It may be the result of paralysis, paralytic or intention tremor; of poisoning, toxic tremor; of some functional nervous disease, as the hysterical tremor; or spasm of the muscle, spasmodic or passive tremor; or it may occur as a family peculiarity without any discoverable cause, hereditary or idiopathic tremor.

Tremors are also classified as rapid, in which the movements occur more than five times per second; and slow, in which the oscillations may occur at intervals of several seconds. Nearly all forms of tremor are increased by placing the muscles upon a stretch. Tremor can usually be recognized by simple inspection. Ordinarily it is sufficient, in order to detect tremor of the fingers, to have the patient extend them forcibly and keep them in that position.

Tremors are nearly always exaggerated by fatigue, weakness, excitement, or extreme cold. They develop in most normal persons in extreme forms of any of these states. These tremors are usually coarse, irregular, and either increased by or only apparent upon voluntary movements. Senile tremors are fine tremors, usually of the head and hands. Persistent coarse tremors occur particularly in paralysis agitans. In this the movements in the fingers are those of flexion and extension, and in the thumb of opposition, and it is therefore spoken of as pill-rollers' tremor. Voluntary effort causes it to cease for a brief interval. Tremor occurs also not infrequently in exophthalmic goitre, and is increased by excitement or effort. The hereditary or idiopathic tremor becomes more apparent with advancing age, and is always increased by emotional disturbance. Irregular tremors occur as a manifestation of ataxia; often with cerebral lesions (the paralytic tremor); and after intoxications, as alcohol and tobacco. The hysterical tremor may be either irregular or regular. Its character is largely influenced by surrounding circumstances. Ordinarily, the hysterical tremor, being the result of voluntary and variable effort, is irregular. Intention tremor occurs particularly in multiple sclerosis. In this condition no tremor is observed while the parts are at rest, but as soon as voluntary motion is attempted a violent oscillation ensues, and continues until the effort ceases. Such a tremor can be particularly well elicited by asking the patient to convey a glass of water to his mouth. It may also be tested by asking the patient to touch some object with his forefinger. It

will be observed as the finger approaches, that the oscillations become more vigorous and wider. Intention tremor may, of course, be present in other parts of the body. In some respects it resembles the ataxic tremor.

Other tremors include the tremors of the head, which may be either forward or backward, lateral or rotary, rapid or slow. A peculiar tremor-like movement is the salaam tremor, a slow backward and forward movement that occurs in some forms of idiocy.

FIBRILLARY TWITCHINGS.—These occur in muscles undergoing degenerative changes. They are characterized by the sudden spasmodic contraction of individual fibers in the mass of the muscle itself, giving rise to a curious trembling of the underlying skin. They often occur spontaneously, and in degenerating muscles may be elicited by slight mechanical stimuli, such as cold, percussion, or shock. Fibrillary twitching may also occur in healthy muscles that have either been chilled (tremor or shivering) or subjected to severe fatigue.

CHOREA AND ATHETOSIS.—The typical movement of chorea is an irregular innervation of groups of muscles that appears to be voluntary in character, yet is not under the control of the patient, is much more rapid, as a rule, than a voluntary movement, and recurs at very frequent intervals. Choreic movements may be mild, or so severe that they produce irregular contortions of the body, causing the patient to throw himself or herself from side to side and thus producing severe bodily injuries and even death by exhaustion. *Athetosis* is a name given to a peculiar slow, irregularly rhythmical movement of the extremities, generally spoken of as worm-like in character. It is ordinarily most marked in the fingers. The limbs may be affected, giving rise to a curious staggering gait in which the patient seems ever to be about to lose his equilibrium, but maintains it almost like a miracle. Frequently the muscles of the face are involved, producing curious irregular grimaces and more or less disturbance of speech or dysarthria. The movements are usually continuous. Athetosis is a very common sequel to cerebral lesions occurring in early childhood.

Tic.—Habitual twitching of a muscle or group of muscles is known as "tic," and is a purely functional condition. *Tic convulsif*, or general tic, and *facial tic* are the forms of tic usually noted.

The Reflexes.—These consist essentially of involuntary contraction of a muscle in response to a peripheral stimulus. They depend upon the integrity of the so-called reflex arc, which consists of (1) an afferent (sensory) nerve which conveys the impulse through the posterior root ganglia into the spinal cord; (2) the motor cells of the anterior cornua from which, in response to the arriving stimulus, an impulse is sent out through (3) an efferent (motor) nerve to the muscle, causing its contraction. The reflex arc, furthermore, is in some way coördinated or inhibited by fibers passing from the cerebrum or cerebellum, through the pyramids to the anterior horn cells, though the integrity of the reflex arc is necessary for the performance of reflex acts. Thus

lesions anywhere in the reflex arc will lessen or abolish the reflexes; lesions in the motor tracts in the lateral columns will exaggerate the reflexes, the result of abolition or lessening of the inhibitory impulses from these tracts.

Three varieties of reflexes are recognized:

- A. Deep or tendon reflexes.
- B. Superficial or cutaneous reflexes.
- C. Visceral reflexes.

A. Deep Reflexes.—These are elicited by striking a tendon of a muscle near its insertion, causing contraction of the muscle. The various deep reflexes are numerous and such reflexes are frequently of little clinical importance, as they are usually inconstant and uncertain. The following deep reflexes are of diagnostic value:

1. **REFLEXES OF THE HEAD.**—The most important reflexes of the head include the *supra-orbital reflex*, which consists of a slight contraction of the eyelid when the supra-orbital nerve is struck. It is absent in lesions of the supra-orbital nerve and in facial palsy. The *malar reflex* is elicited by striking upon the malar bone causing elevation of the angle of the mouth. The *chin-jerk* is elicited by percussion upon a finger laid across the lower teeth. Under normal conditions there will be a slight upward jerk of the chin. The chin-jerk is increased in hysteria and in cachectic and comatose conditions.

2. **REFLEXES OF THE UPPER EXTREMITY.**—The *biceps reflex* is elicited by tapping upon the tendon in the bend of the elbow when the arm is semiflexed. The *triceps reflex* is elicited by striking upon the tendon over the olecranon process. In testing these two reflexes the arm of the patient should be supported upon the forearm of the examiner in order to relax the muscles of the patient.

The *wrist-jerk* is elicited by fixing the hand as in wrist-drop and striking the extensor tendons near the wrist-joint, when an upward jerk of the hand should occur. The *scapulohumeral reflex* is elicited by tapping upon the spinal border of the scapula just above the inferior angle. This normally causes an external rotation of the arm. In disease of the pyramidal tracts above the cervical enlargement, this reflex is not only exaggerated, but, in addition, the shoulder is lifted, the arm thrown from the side, the forearm flexed upon the arm, and the fingers extended.

3. **REFLEXES OF THE LOWER EXTREMITY.**—These reflexes are the most important of all. The *knee-jerk*, or *patellar tendon reflex*, is invariably present in health, and by its delicacy and constancy is the most valuable reflex for clinical purposes. It may be elicited in a variety of ways. The patient may lie upon his back; then placing one hand under the knee it should be lifted several inches from the surface of the bed or table. Then with the ulnar side of the hand or the percussion hammer, the patellar tendon is struck a sharp blow between the lower edge of the patella and the tubercle of the tibia. The most obvious and vigorous contraction occurs in the quadriceps of the same side,

causing the leg to be tipped upward suddenly and giving rise to the name knee-jerk. In addition, the adductors of the same side nearly always contract slightly, and occasionally the flexor muscles also contract. Another method of obtaining this reflex is to allow the patient to sit on a low chair with the leg extended forward until it forms a blunt angle with the thigh, the heel being rested upon the ground. The patellar tendon is then struck as before. Clinically, it is usually sufficient when the patient is sitting in an ordinary chair, to have one leg thrown over the other and hanging loosely and freely. Exaggeration of the knee-jerk is characterized by a more vigorous effort or more extensive contraction of the surrounding muscles. The patellar tendon reflex is said to be invariably present in health, but its intensity varies considerably, and in some apparently healthy persons without any evidence of disease of the spinal cord, it is extremely difficult to elicit.

FIG. 27



Testing the knee-jerk.

Under these circumstances it is necessary to use various procedures in order to make it evident. These consist either in requesting the patient to look at the ceiling, in order to distract the attention, or to perform some violent muscular effort, such as an attempt to pull the hands apart when they are clasped together, to squeeze the dynamometer, etc. Under these circumstances the knee-jerk, if obtained, is spoken of as *reinforced* (Jendrassik's method). It is always important to have the muscles completely relaxed, and to persuade the patient not to think of what is being done. The knee-jerk is sometimes rendered more pronounced by emotion, and sometimes inhibited, as by fright. *Patellar clonus* occasionally occurs, and is obtained by placing the thumb and forefinger on the upper edge of the patella, and pushing it forcibly downward and keeping it in that situation. If clonus occurs, it will be characterized by a series of rapid contractions of

the quadriceps, resulting in a vertical oscillation of the patella. It occurs in disease of the spinal cord, and not infrequently in conditions of increased tonicity in general infectious diseases.

Next in importance to the knee-jerk is the *Achilles tendon reflex*, which consists in the contraction of the gastrocnemius and soleus muscles when the Achilles tendon is struck. It is most readily elicited by lifting the entire leg from the bed or table, and holding it by the ball of the foot, which is gently pressed upward. The tendon is thus moderately stretched, and may be struck directly. In nearly all healthy individuals this reflex is present, but is absent in some, and its absence is apparently of no clinical significance. Exaggeration may be indicated in moderate cases by the more forcible extension of the foot. In more pronounced cases it gives rise to a peculiar and characteristic phenomena known as *ankle-clonus*. This is readily

FIG. 28



Ankle-clonus.

produced by slightly flexing the leg and the thigh, then grasping the ball of the foot firmly, flexing it dorsally with considerable force, and holding it in that position. When ankle-clonus exists, there will be violent vibratory oscillations of the foot as long as the pressure upon the sole is continued, that vary from two or three up to five or ten movements per second. Occasionally, in very mild cases, the clonus after a few movements becomes weaker and rapidly disappears. Ankle-clonus is present in any organic condition of exaggerated reflexes. A pseudo-ankle-clonus has been described as characterized by a few irregular oscillations that soon cease. It occurs in functional disease and occasionally among malingerers.

The other reflexes of the lower extremities are *front tap*, dorsal extension of the toes upon percussion of the anterior surface of the tibia and *Sinkler's reflex*, which is elicited by flexing the great toe upon the sole. The foot is then dorsally flexed, the leg flexed on the thigh, and the thigh on the abdomen, so that the limb is drawn up. It occurs in conditions that cause extreme spasticity of the limbs, such as transverse myelitis.

DIAGNOSTIC SIGNIFICANCE OF THE DEEP REFLEXES.—I. *Diminished or Absent Reflexes.*—1. Disease of the peripheral nerves, a polyneuritis, or more rarely a localized neuritis causing loss of reflexes in the muscle supplied by the affected nerve.

2. Disease of the posterior nerve roots and columns: (a) tabes dorsalis, (b) Friedreich's ataxia.

3. Disease of the anterior cornua: (a) anterior poliomyelitis; (b) Landry's paralysis; (c) progressive spinal muscular atrophy.

4. Miscellaneous disease: (a) complete transverse lesions of the cord in some cases; (b) spinal meningitis; (c) apoplexy immediately after the cerebral insult; (d) epilepsy immediately after the convulsion.

II. *Exaggeration of the Reflexes.*—1. Intracranial lesions: (a) hemorrhage, thrombosis, and embolism; (b) birth palsies; (c) tumor or abscess; (d) Marie's cerebellar hereditary ataxia; (e) general paralysis of the insane.

2. Disease of the cord (lateral columns): (a) amyotrophic lateral sclerosis; (b) multiple sclerosis; (c) lateral sclerosis (spastic paraplegia); (d) lateral sclerosis associated with other cord conditions, syringomyelia, multiple sclerosis, and ataxic paraplegia.

3. Transverse lesions of the cord above the segments controlling the reflexes: (a) external compression by tumors, caries of vertebrae, etc.; (b) internal compression by hemorrhage; (c) myelitis.

4. Functional disorders: (a) hysteria; (b) neurasthenia, etc.

5. Intoxications: (a) tetanus; (b) strychnine-poisoning; (c) hydrophobia.

III. Superficial or Cutaneous Reflexes.—These reflexes, though inconstant both in health and disease, are of value in localizing the level of special lesions, showing integrity of the reflex arc when present and in indicating lesions of the upper motor neuron when exaggerated. They are elicited by stroking or irritating the skin, and are due to the contractions of voluntary muscle beneath the skin near the point of irritation. The more important are:

Plantar reflex, obtained by tickling the sole of the foot so that the hip and the knees are suddenly flexed and the toes plantar flexed, is probably the most important of the superficial reflexes and depends upon the integrity of the pyramidal tracts. When these tracts are diseased a converse dorsal flexion of the toes occurs, the *Babinski phenomenon*, which is best elicited by stroking the sole of the foot from the heel toward the toes. It is also usually seen in infants. The *paradoxical reflex*, dorsiflexion of the big toe, elicited by pressure

upon the calf muscle and *Oppenheim's reflex*, dorsiflexion of the big toe upon forcibly pressing along the inner border of the tibia, are also usually significant of disease of the lateral columns.

The *cremasteric reflex*, retraction of the testicle upon stroking the inner side of the thigh of the same side, and the *gluteal reflex*, dimpling of the gluteal fold on stroking the buttocks, are fairly constant reflexes.

FIG. 29



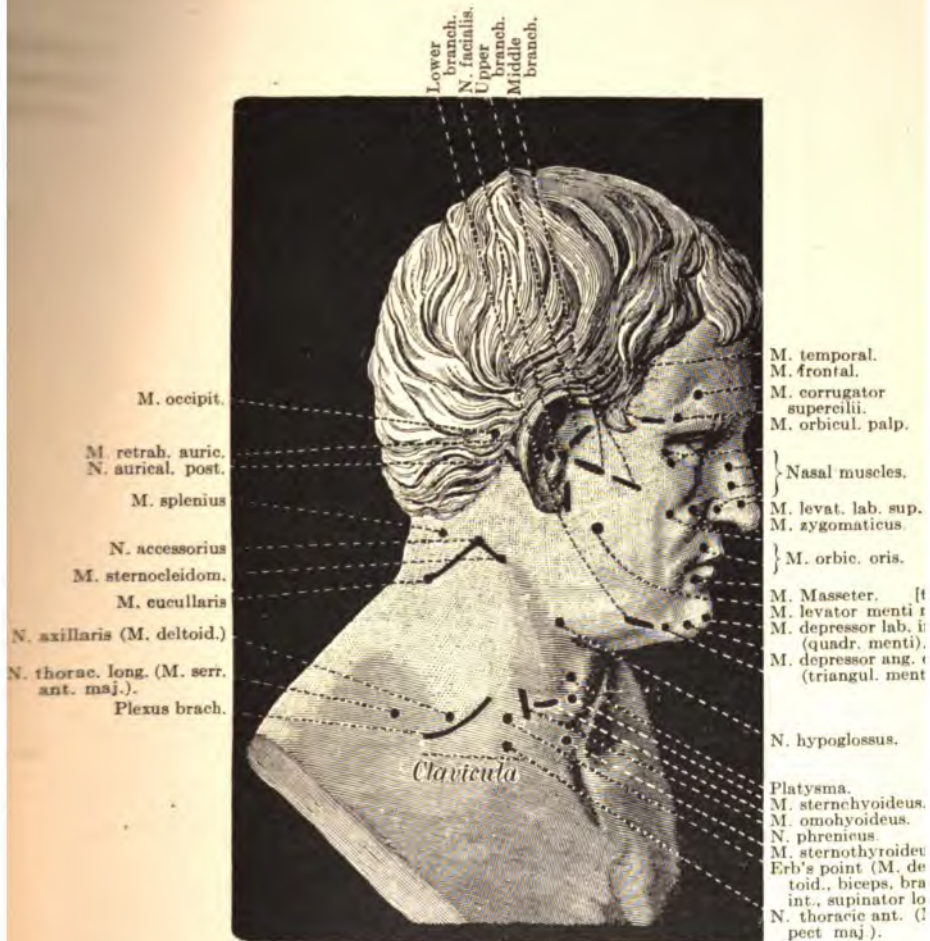
Testing the Babinski reflex.

The *abdominal reflexes* (epigastric, umbilical, or hypogastric) are elicited by stroking the abdomen, causing contractions of the recti and oblique muscles. The *scapular reflex* consists of contraction of the scapular muscles upon irritating the intrascapular region. The *corneal reflex*, closure of eyelid upon touching the cornea, depends upon the integrity of the fifth nerve. The *palatal reflex*, elevation of the soft palate when touched, and the *pharyngeal reflex*, choking and coughing upon touching the pharyngeal wall, depend upon the integrity of the ninth, tenth, and eleventh cervical nerves, and are consequently abolished in bulbar palsy.

C. Visceral Reflexes.—These are complicated reflexes, of which the bladder and rectal reflex are of the most diagnostic importance. In general, disease of the spinal cord above the fourth and fifth sacral segments causes loss of control by the higher inhibiting centres; disease of the centres in these segments causes abolition of the reflex arc and a consequent inability to control the excretion of urine and feces.

power, or change in consistency or tone are present. A method of determining the nutritional state of the muscle is by electrical current, which is described in the succeeding section.

FIG. 30

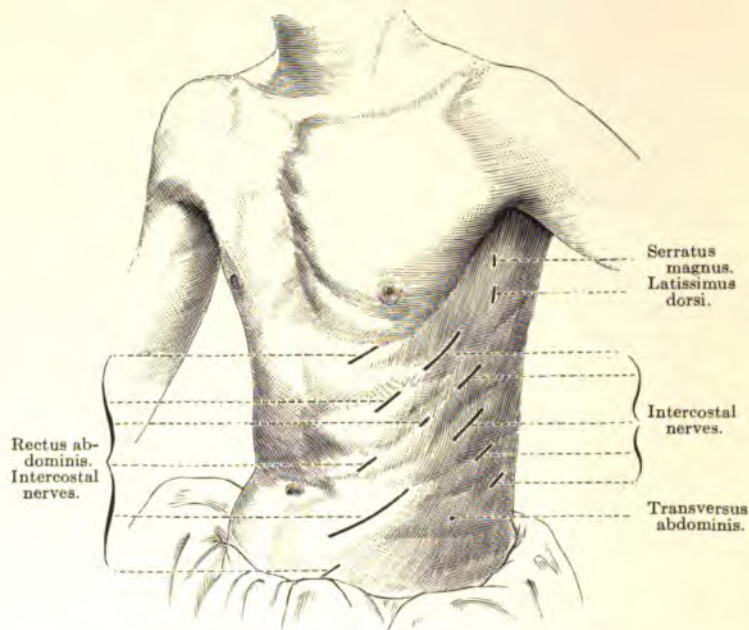


Motor points for the head and neck. (Sahli.)

Electrodiagnosis.—In electrodiagnosis we use two types of apparatus, the galvanic and the faradic, to study the contractility of the muscle. With the *galvanic* apparatus the contraction occurs only when the current is made or broken. The galvanic current is produced by the galvanic battery, to which long wires are attached. The free end of the wire toward which the current flows from the cell is called the *anode*, or positive pole, and the free end from which the current passes

to the cell the *cathode*, or negative pole. It is customary to introduce a galvanometer graduated in milliampères into the current for measuring the amount of electricity employed. The apparatus is also provided with a rheostat, which renders it possible, by the introduction of a greater or less degree of resistance, to regulate the amount of electricity passing through the body. The free ends of the wires are supplied with electrodes, the one large, the other small, which are covered with cotton and moistened when about to be used. If a muscle or nerve is to be investigated, the large electrode is placed over the back or the sternum.

FIG. 31

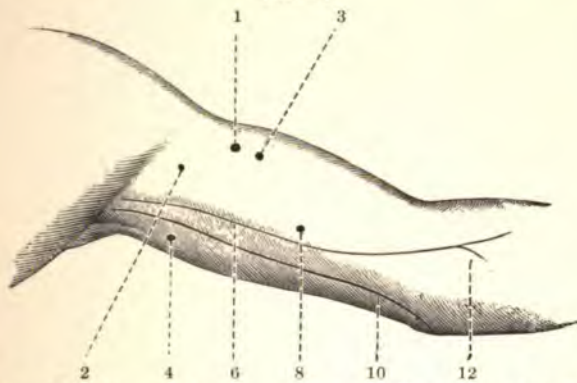


Motor points of the trunk. (From von Ziemssen.)

The current is so arranged that this large electrode is at first the anode and the small electrode the cathode. The cathode is now placed over the muscle or the nerve to be stimulated, locating it, if possible, exactly over the most sensitive (electrically) point. (See Figs. 30 to 39.) The circuit should be opened and the rheostat so placed that the minimum amount of current flows through the body. The current is gradually increased by moving the rheostat, and the current rapidly opened and closed until a slight twitching of the muscle appears upon the instant of closing the circuit, the cathodal closing contraction or CCC. The current should now be slightly increased, and by means of a switch the small electrode converted into the anode and the other into the cathode. Contractions now take place which are spoken of as the

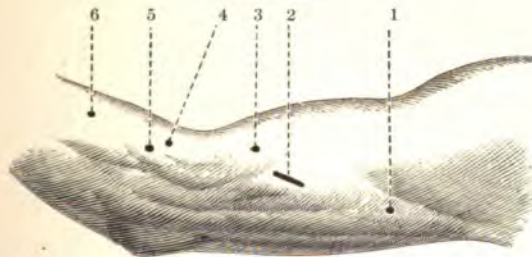
anodal closing contraction, or ACC, and the anodal opening contraction, or AOC. If the small electrode is again made the cathode, it will be found that there is a vigorous contraction when the current is closed, but none when it is opened, unless the current is very strong, when the cathodal opening contraction or COC occurs. The order of reaction of normal nerve and muscle may be designated by the following formula: CCC > AOC > ACC > COC. The symbol > means greater or less than, the apex pointing in the direction of the lesser of the two reactions.

FIG. 32



Motor points of the arm, under side. (From von Ziemssen.) 1, musculocutaneous nerve; 2, musculocutaneous nerve; 3, biceps; 4, internal nerve of the triceps; 6, median nerve; 8, brachialis anticus; 10, ulnar nerve; 12, branch of the median nerve to the pronator teres.

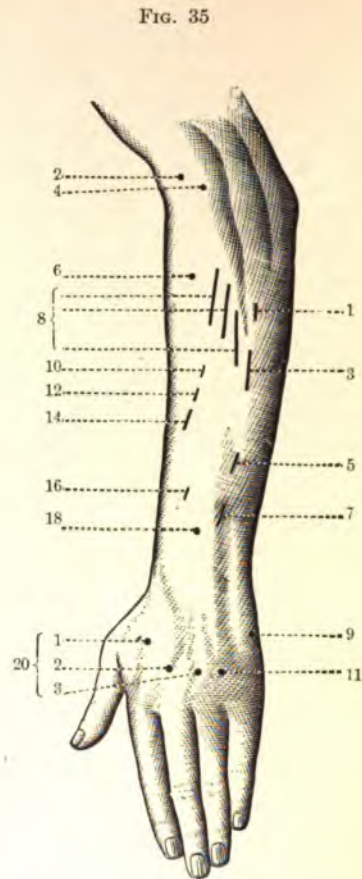
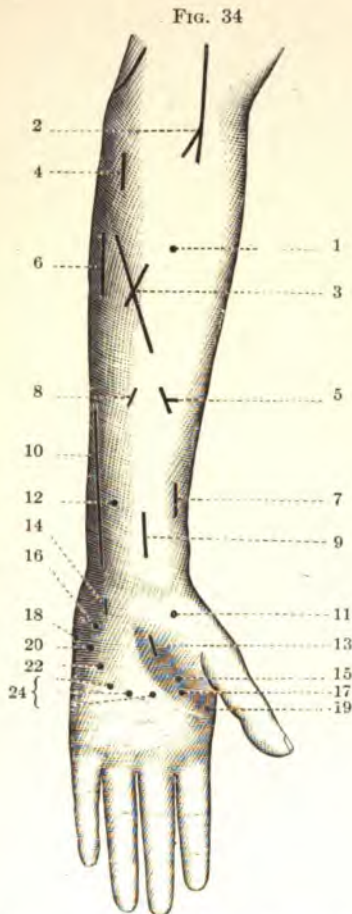
FIG. 33



Motor points of the arm, outer side. (From von Ziemssen.) 1, external head of the triceps; 2, musculospiral nerve; 3, brachialis anticus; 4, supinator longus; 5, extensor carpi radialis longior; 6, extensor carpi radialis brevior.

Under ordinary circumstances the healthy muscle contracts suddenly and relaxes almost immediately. Various modifications of these phenomena occur in diseased conditions, and there are considerable quantitative changes in the different muscles in health. Thus in the muscles of the face contraction is always more rapid than in those of the thigh, and can be elicited with much weaker currents. In disease we recognize two types of alteration: (1) quantitative changes; (2) qualitative changes.

The *faradic apparatus* produces induced currents. The strength of the current is modified by the position of the outer and inner coils,



Motor points of the forearm, inner surface. Motor points of the forearm, outer surface.
(From von Ziemssen.)

FIG. 34.—1, flexor carpi radialis; 2, branch of the median nerve for the pronator teres; 3, flexor profundus digitorum; 4, palmaris longus; 5, flexor sublimis digitorum; 6, flexor carpi ulnaris; 7, flexor longus pollicis; 8, flexor sublimis digitorum (middle and ring fingers); 9, median nerve; 10, ulnar nerve; 11, abductor pollicis; 12, flexor sublimis digitorum (index and little fingers); 13, opponens pollicis; 14, deep branch of the ulnar nerve; 15, flexor brevis pollicis; 16, palmaris brevis; 17, adductor pollicis; 18, adductor minimi digiti; 19, lumbricalis (first); 20, flexor brevis minimi digiti; 22, opponens minimi digiti; 24, lumbricales (second, third, and fourth).

FIG. 35.—1, extensor carpi ulnaris; 2, supinator longus; 3, extensor minimi digiti; 4, extensor carpi radialis longior; 5, extensor indicis; 6, extensor carpi radialis brevior; 7, extensor secundi internodii pollicis; 8, extensor communis digitorum; 9, abductor minimi digiti; 10, extensor indicis; 11, dorsal interosseus (fourth); 12, extensor indicis and extensor ossis metacarpi pollicis; 14, extensor ossis metacarpi pollicis; 16, extensor primi internodii pollicis; 18, flexor longus pollicis; 20, dorsal interossei.

which is spoken of as the coil distance and measured in centimeters. The electrodes are placed as with the galvanic apparatus and the current gradually increased until contractions of the muscle ensues. It will be noted that contraction of muscle occurs in the region of the distal electrode, irrespective of which electrode has been employed,

FIG. 36

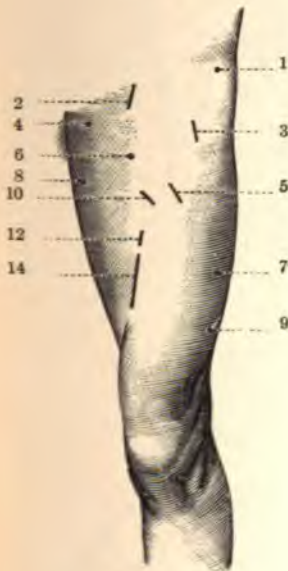
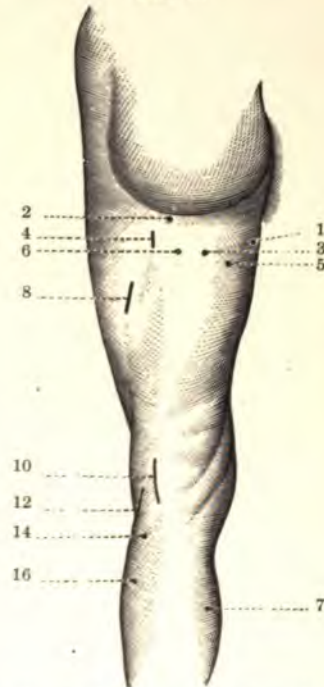


FIG. 37



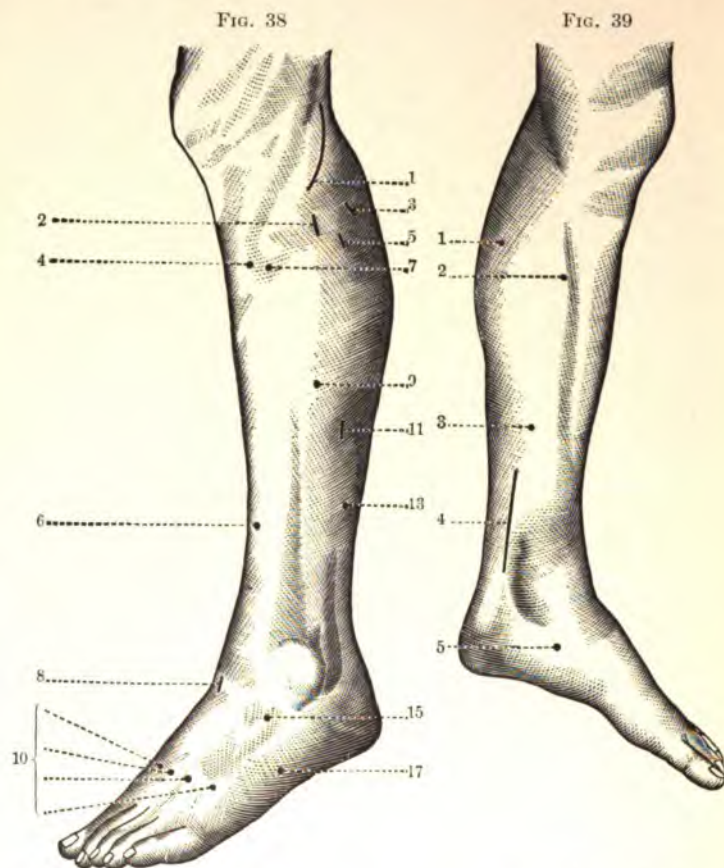
Motor points of the thigh, anterior surface. Motor points of the thigh, posterior surface.
(From von Ziemssen.)

FIG. 36.—1, tensor vaginae femoris (branch of the superior gluteal nerve); 2, anterior crural nerve; 3, tensor vaginae femoris (branch of the crural nerve); 4, obturator nerve; 5, rectus femoris; 6, sartorius; 7, vastus externus; 8, adductor longus; 9, vastus externus; 10, branch of the crural nerve to the quadriceps extensor cruris; 12, crureus; 14, branch of the crural nerve to the vastus externus.

FIG. 37.—1, adductor magnus; 2, inferior gluteal nerve for the gluteus maximus; 3, semitendinosus; 4, great sciatic nerve; 5, semimembranosus; 6, long head of the biceps; 7, gastrocnemius (internal head); 8, short head of the biceps; 10, posterior tibial nerve; 12, peroneal nerve; 14, gastrocnemius (external head); 16, soleus.

upon opening the circuit and continues as long as the current is maintained. Furthermore the stronger the current the more pronounced the contractions. If the muscles are atrophied or if they be paralyzed as a result of disease of the lower motor neuron, the response to the faradic current is diminished or absent. In acute anterior poliomyelitis and other diseases inducing rapid muscular degeneration,

the diminution of contractility to the faradic current is a guide to the extent of paralysis and atrophy that may ensue.



Motor points of the leg, outer side.

Motor points of the leg, inner side.

(From von Ziemssen.)

FIG. 38.—1, peroneal nerve; 2, peroneus longus; 3, gastrocnemius (external head); 4, tibialis anticus; 5, soleus; 6, extensor longus pollicis; 7, extensor communis digitorum longus; 8, branch of the peroneal nerve for the extensor brevis digitorum; 9, peroneal brevis; 10, dorsal interossei; 11, soleus; 13, flexor longus pollicis; 15, extensor brevis digitorum; 17, abductor minimi digiti.

FIG. 39.—1, gastrocnemius (internal head); 2, soleus; 3, flexor communis digitorum longus; 4, posterior tibial nerve; 5, abductor pollicis.

Alterations in the Reactions of the Muscles and Nerves to Electricity.—**REACTIONS OF DEGENERATION.**—Quantitative alterations consist in increase or decrease of the susceptibility of the muscles or nerves to electrical action. Quantitative increase in the electrical reaction occurs chiefly in tetany, for which disease it is almost pathognomonic,

and has been spoken of as Erb's sign. It occurs also occasionally in the early stages of peripheral palsies, and occasionally in tabes and hemiplegia. Diminished electrical irritability occurs in diseases of the lower motor neurons and in atrophies secondary to disease of the joints and loss of functional activity on the part of the muscle.

The *quantitative qualitative reaction* consists in diminution of the reaction of the muscle and nerve to the faradic current and diminution or exaggeration to the galvanic current, with distinct alteration of the order in which the various forms of galvanic irritation produce contractions. These qualitative alterations of galvanic irritability consist in the anodal closing contractions becoming greater than the cathodal closure contractions or $ACC > CCC$ and the cathodal opening contractions equals or is greater than the anodal opening contractions or $COC = \text{or} > ACC$. Thus in a peripheral paralysis during the first week there is diminished quantitative response to both faradic and galvanic currents, and faradic irritability soon disappears altogether. In about two weeks there appears increased quantitative response to the galvanic current and the qualitative reactions mentioned above make their appearance. If recovery ensues the qualitative electrical reactions gradually show improvement and eventually become normal. The abnormal increased galvanic irritability persists for a longer time, but gradually disappears as the faradic irritability returns. If recovery does not take place, all reactions gradually disappear, though the increased galvanic irritability may last for months. The sluggish inactive reactions of degenerating muscle to galvanic currents is extremely typical.

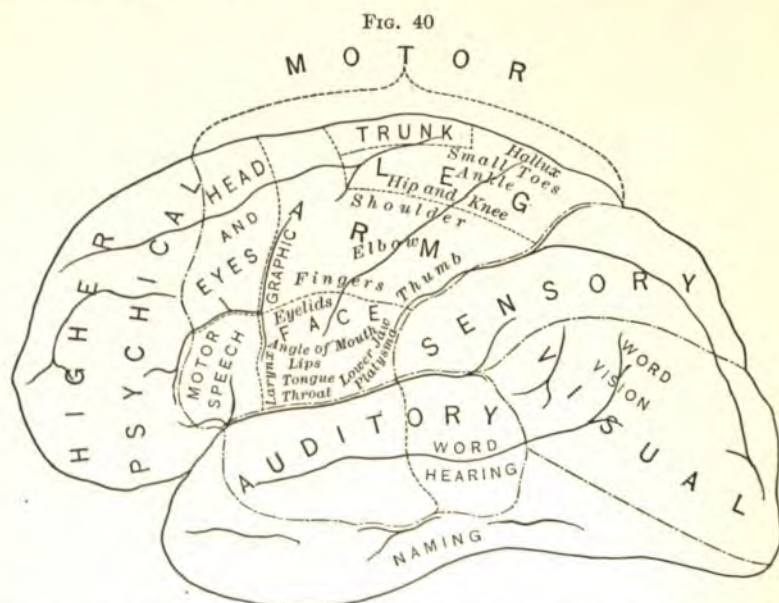
DIAGNOSTIC SIGNIFICANCE OF REACTIONS OF DEGENERATION.—

Quantitative and qualitative reactions of degeneration occur primarily as the result of disease of the peripheral motor neuron. They are, therefore, found in all diseases of the spinal cord that affect the anterior cornua or the motor roots, and in all diseases of the medulla that affect the motor nuclei or their roots; in acute and chronic anterior poliomyelitis, progressive spinal muscular atrophy, and in bulbar palsy, in transverse myelitis, syringomyelia, tumor of the cord, and as a result of chronic forms of meningitis, or disease of the vertebral column pressing upon the roots. They are also found in all forms of peripheral neuritis, whether toxic, infectious, or traumatic, and in all cases of solution of continuity of the nerves. They occur in the so-called idiopathic muscular dystrophies, but in these they are exceptional. They are also found in a few cases after cerebral lesions.

The reactions of degeneration may be used for determining the prognosis of the case. When after the sixth week the muscle does not respond as readily as before to direct galvanic stimulation, and the cathodal closing contraction becomes equal to or greater than the anodal opening contraction, the prognosis is exceedingly favorable; the increased rapidity of the contraction particularly is of great significance. If, on the other hand, after from six to twelve weeks no change has

occurred, the anodal still precedes the cathodal contraction, and both are worm-like in character, the prognosis is doubtful. Months, however, may elapse before the muscle gradually begins to regain its normal character.

ATYPICAL FORMS OF THE REACTIONS OF DEGENERATION.—Only two of these are important. In the *myotonic* reaction the muscular contraction persists after the electrical stimulus has been removed. This occurs both with the faradic and the galvanic current, but the order of contraction of the various forms of stimulation of the latter is not altered. Myotonic reaction is pathognomonic of Thomsen's disease—myotonia congenita. The *myesthetic* reaction is characterized by the rapid exhaustion of the muscle or nerve, so that relaxation may take place while the faradic current is still being employed. It occurs in the various types of myasthenia.



Cortical centres and areas of representation on the lateral aspect of the hemispheres. (Mills.)

Disorders of Nutrition.—**Trophic Changes in the Skin and Subcutaneous Tissues.**—Disorders of nutrition or trophic changes are lesions produced in the tissues as a result of defective or altered innervation. (See Chapter XIII, Examination of the Skin.)

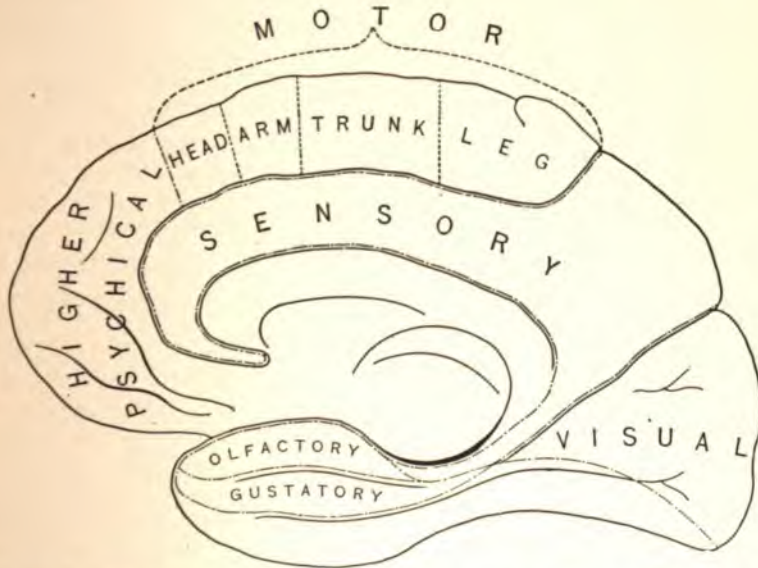
Trophic Changes in the Bones and Joints.—(See Chapter XV, Examination of the Bones and Joints.)

The Contour of the Body.—Alteration of the contour of the body occurs in various nervous diseases. (See Chapters IX, X, and XI.)

Changes in the Extremities.—(See Chapter XII.)

Localization of Cortical Lesions.—The cortex of the brain has been divided into various regions which are referred to certain fissures. The most important of these is the fissure of Sylvius. Around its posterior extremity winds the convolution known as the gyrus angularis. Next is the Rolandic fissure, passing from the superior longitudinal fissure to the fissure of Sylvius, with which it forms an acute angle. These two contain nearly all the motor centres. The third prominent fissure is the occipitoparietal, separating the parietal from the occipital lobe. On the median surface it unites at an acute angle with the calcarine fissure, the two enclosing between them the triangular convolution

FIG. 41



Cortical centres and areas of representation on the mesial aspect of the hemisphere.
(Mills.)

known as the cuneus (Fig. 40). The motor centres are so arranged that those for the face are in the lowest portion of the motor region, those for the arms just above them, those for the legs above these, and those for the trunk in the posterior termination of the ascending parietal convolution, along the margin of the superior longitudinal fissure. These centres do not represent particular muscles, but particular forms of movement, involving frequently the simultaneous contraction of several muscle groups. The motor region for speech, discovered by Broca, occupies the posterior portion of the third frontal convolution and the lower part of the ascending frontal convolution. The terminations of the sensory neurons have not yet been conclusively

determined. It seems likely that some of them terminate in the motor region and others in the upper portion of the parietal lobe.

However, it is known that the cuneus of the median surface of the occipital lobe appears to receive directly the fibers from the optic tract; that the centre for audition is situated in the temporosphenoidal convolution; and that the centres for smell and taste are in the uncinate and fornicate convolutions. The functions of the frontal lobes are not well known. It is supposed that they are the seat of intelligence.

The functions of the basal ganglia of the brain are as yet insufficiently known to enable us to diagnosticate lesions situated in them with certainty. Lesions in the lenticular nucleus may be latent. In some cases they appear to have produced sensory disturbances. The *optic thalamus* appears to receive fibers from many parts of the cortex, and lesions in this region frequently produce sensory disturbances. The *pulvinar* appears to be one of the three basal ganglia associated with the optic tract, and when it is destroyed there is usually bilateral or contralateral hemianopsia. The existence of mimic paralysis has, in a few cases, led to the correct diagnosis of thalamic lesions. The *anterior corpora quadrigemina* are apparently the situations in which the arch of the pupillary reflex is completed. The *internal geniculate ganglia* and the *posterior corpora quadrigemina* appear to be associated with hearing.

Lesions in the *pons* and *medulla* produce, as a rule, characteristic symptoms that make it possible to locate them with considerable accuracy, due to the fact that the nuclei of nine cranial nerves are situated in these two portions of the brain. The nucleus of the oculomotor nerves is found surrounding the anterior portion of the aqueduct of Sylvius, just beneath the anterior corpora quadrigemina. Destructive lesions cause partial or complete ophthalmoplegia, and there is, therefore, abolition of the pupillary reflex. Just behind the nucleus of the third nerve, and beneath the posterior quadrigemina, is a small group of cells for the pathetic nerve. The nucleus of the trigeminus is situated in the anterior portion of the pons, just to the outer side of the fillet, the motor group of cells lying inside the sensory group. The Gasserian ganglion receives the peripheral branches of this nerve and correspond to the spinal ganglia. Disturbances of the nucleus produce anesthesia on the same side of the face, including the conjunctiva and the mucous membrane of the mouth. There is loss of taste in the anterior two-thirds of the tongue, and there is some disturbance of smell in the nostril on the same side. Mastication is imperfect. Irritative lesions cause tic douloureux. This may also be the result of disease of the ganglion. The nucleus of the *abducens* lies in the posterior portion of the pons, just beneath the floor of the fourth ventricle. Destructive lesions cause internal strabismus. The nucleus of the *facial nerve* is found in the posterior portion of the pons, lying lightly behind and to the median side of the nuclei for the trigeminus. Destructive lesions

cause paralysis of the same side of the face, usually involving the upper branch. (See Hemiplegia.) Irritative lesions cause facial tic. The nucleus of the *acusticus* is found in the anterior portion of the medulla oblongata, just beneath the floor of the fourth ventricle, lying immediately above the superior olivary body. Lesions produce nerve or mental deafness on the same side. The nuclei of the *vagus* and the *glossopharyngeal* nerves are apparently in the jugular and petrosal ganglia. From these ganglia fibers pass into the medulla oblongata at its lateral aspect, and end in a nucleus in the floor of the fourth ventricle. Close to the median line is the *hypoglossal* nucleus. Its destruction produces paralysis and degenerative atrophy of the corresponding side of the tongue.

The functions of the *pons* are merely those of the centres and tracts it contains. On account of the decussation of the central fibers for the facial nerve in this region, crossed paralysis is usually considered pathognomonic of pontine disease. The functions of the *medulla* are also largely dependent upon the nuclei and tracts it contains. As it contains the centres for the pneumogastric and some of the centres or tracts of fibers for respiration, lesions in it are usually very promptly followed by death. Lesions of the *restiform bodies* are frequently associated with nystagmus and may cause the symptoms of cerebellar ataxia. As the medulla contains the nuclei of the motor nerves to the pharynx, larynx, and mouth, paralysis of the muscles of this region is spoken of as bulbar palsy.

The *cerebellum* is supposed to be concerned in coördination and the maintenance of the equilibrium. The hemispheres may, however, be extensively diseased without giving rise to any symptoms. If the middle lobe is affected, the characteristic manifestations are disturbance of equilibrium and incoördination. The gait resembles that of a drunken man. Nystagmus is frequent, especially in cases of tumor, and choked disks appear early. If the medullary peduncle is affected by an irritative lesion quite characteristic symptoms result. There are forced movements, that is to say, the patient may have an irresistible tendency to fall toward or lie upon the side. There are no symptoms diagnostic of disease of the superior or middle peduncles. Disease of one side of the pons may cause symptoms similar to those of cerebellar trouble.

General Symptomatology of Lesions of the Brain.—Lesions of the brain may be irritative or destructive. The former, if affecting the motor tract, produce clonic spasms. If destructive, they produce paralysis without atrophy, and cause increase in the muscle tone by removal of the influence of the superior arc and exaggeration of the reflexes. All these changes occur in the muscles of the opposite side of the body. Irritative lesions are most likely to be extracerebral—that is, pressing upon the cortex. Lesions in the brain substance are usually destructive, and therefore cause paralysis. As motor fibers are distributed over a considerable area of the cortex, lesions in this

region, if circumscribed, are likely to cause at least monoplegia. If they involve the area for the face, the upper branch of the facial nerve, which is innervated from both sides, is rarely affected. Aphasia occurs only when the left side is diseased. Lesions in the corona radiata near the cortex usually cause monoplegia; if the lesion is near the internal capsule, hemiplegia is more common. Lesions in the internal capsule almost invariably cause hemiplegia. If the knee and anterior portion of the posterior limb are involved, hemiplegia without sensory changes results. When the posterior third of the posterior limb is involved, sensory disturbances are present, and there is likely to be hemianopsia. Lesions in the anterior portion of the anterior limb produce no recognizable symptoms and are termed latent. Increase in intracranial pressure may be brought about by newgrowths, traumatism, edema, or inflammation. There are usually headache, delirium or coma, vomiting, and choked disk. If the process is of slow development, a certain amount of adaptation may occur, and only the headache and vomiting may be present.

Localization of Spinal Lesions.—The spinal cord may be regarded in two ways: (1) as the pathway between the peripheral nervous system and the brain, containing the tracts running from the brain to the motor nerves, and from the sensory nerves to the brain; (2) as a number of groups of ganglion cells arranged in horizontal layers or segments. These segments are usually classified according to the nerve roots that spring from them. There are, therefore, eight cervical, twelve dorsal, five lumbar, and five sacral segments of the cord. The white matter of the spinal cord is divided into two regions: the anterolateral part, extending from the median fissure to the posterior horns, and the posterior part, lying between the posterior horns. The anterolateral part contains the motor fibers or pyramidal tracts, whose functions have already been described. In addition, there are certain fibers that pass downward, the functions of which are not certainly known. The gray matter of the cord is divided into the anterior and the posterior horns. It is composed of nerve cells and nerve fibers. The nerve cells in the anterior horns form a large group, which send their axicylinders into the anterior roots, and comprise the peripheral motor neurons. In the posterior horns in the dorsal region there is a group of cells on the inner side known as the column of Clarke, which apparently has something to do with equilibrium. The gray matter also contains a large number of nerve fibers, some of which pass transversely and apparently are concerned in reflex action; others ascend, and convey to the brain the sensations of pain, heat, and cold. Each segment of the cord innervates and receives sensory impressions from an approximately corresponding segment of the body, and contains the lower reflex arcs. The motor and reflex functions of the various segments are shown in the table and the sensory functions in Plate III.

TABLE OF MOTOR AND REFLEX FUNCTIONS OF THE SEGMENTS OF THE SPINAL CORD.
MODIFIED FROM GOWERS, MÜLLER, AND WICHMANN

Segments.	Motor innervation.	Reflexes.
C		
1) Small rotators of head	Scaleni. Lev. ang. scapulae. Subscapularis.	Dilatation of the pupil, sensory part (?)
2) Depressors of hyoid		
3) Diaphragm		
4) Platysma (?)		
5) Deltoid	Pronators Triceps Extensors of wrist and fingers	Scapular.
Biceps		
Coracobrachialis		
Supinator longus		
Spinati	Muscles of hand Extensors of thumb	Tendon reflexes of the mus- cles of the arms.
Serratus major		
6) Pectoral. maj. (clav.)		
7) Subscapularis		
Flexors of wrist and fingers	Erectors of spine	Dilatation of pupil, motor part (?)
Pectoralis (costal)		
Latissimus dorsi		
8) Teres major		
D		
1	Erectors of spine	Epigastric.
2		
3		
4		
5		
6) Intercostal muscles		
7		
8		
9		
10) Abdominal muscles		
11		
12		
L		
1 Quadratus lumborum	Quadriceps	Abdominal.
2 Iliopsoas		
Cremaster		
3 Sartorius		
Pectineus	Extensors of foot Tibialis anticus Peroneal muscles Perineal and anal muscles	Cremasteric.
Adductor		
4 Gracilis		
Obturator		
5 Adductors	Achilles tendon reflex Plantar reflex	Knee-jerk.
Flexors of knee		
Gluteal		
Extensors of foot		
Tibialis anticus	Centres for the bladder and rectum.	Gluteal reflex.
Peroneal muscles		
Perineal and anal muscles		
Anal rotators of sph.		

General Symptoms of Disease of the Spinal Cord.—These depend upon the segment of the cord and upon the nerve tracts involved. Lesions are spoken of as transverse if they involve the whole cord, unilateral if they involve but one side, and focal if they involve only a circumscribed portion.

Transverse Lesions of the Spinal Cord.—Transverse lesions may be produced by inflammation or by pressure either of a tumor or as a result of deformity of the vertebral column (Pott's disease). Lesions above the fifth cervical segment usually cause death by paralysis of the diaphragm. If the patient survives, paralysis of all four extremities and total anesthesia of the body remain. There are also paralysis of the bladder and rectum and abolition of the cutaneous reflexes, and in nearly all cases of the tendon reflexes. Transverse lesions between the fifth cervical and the first dorsal segments produce atrophy and degeneration of certain muscles of the arm, according to their situation; spastic paralysis of the legs and total anesthesia of the body as far up as the part that transmits sensation to the lowest intact segment; paralysis of the bladder and rectum; abolition of the reflexes whose arcs are found in the segment involved; and, if the destruction is not complete, exaggeration of all the tendon reflexes that are completed in the lower segments are present. The cutaneous reflexes are abolished. Lesions of the dorsal region produce spastic paraplegia and paralysis of the bladder and rectum. The arms escape and respiration is not disturbed. The anesthesia extends up to the segment involved. Lesions in the lumbar region produce atrophy and disturbance of sensation, distributed according to their extent. The situation of a lesion may be roughly determined by a study of the reflexes. If the lesion involves the segments concerned in any of these, they are of course abolished. If the lesion is above them, they are sometimes exaggerated; if below, they are ordinarily not involved. Lesions of the conus terminalis and the cauda, as they involve a large number of nerve roots, produce a complexity of symptoms. There are irregular areas of anesthesia corresponding to the posterior roots involved, and atrophy and degeneration of the muscles supplied by the anterior roots. The bladder and rectum usually are affected. If the lesion involved only the lowest roots, there is a characteristic saddle-shaped area of anesthesia over the sacrum.

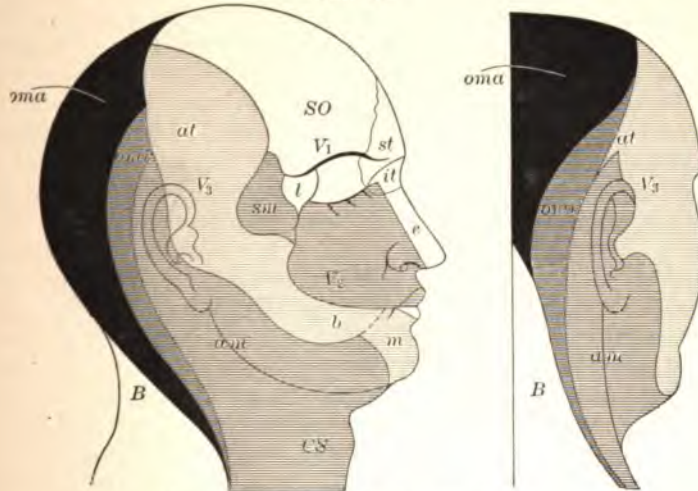
Unilateral Lesion of the Spinal Cord (the syndrome of Brown-Séquard).

This produces paralysis of the same side and anesthesia of the opposite side, both symptoms extending as far upward as the region controlled by the segments that have been affected. Disturbance of sensation is not total. On the side opposite the lesion, tactile, pain, and temperature sense are lost, but the muscular sense persists. On the same side as the lesion there are loss of the muscular sense and complete paralysis. Atrophy and degeneration occur in the muscles supplied by the involved segment; below this there is spastic paralysis, with increase in the reflexes. Above the paralytic area there is a zone of hyperesthesia,

the reason for which has never been satisfactorily explained. The commonest cause of unilateral lesion is traumatism, particularly bullet and stab wounds. Occasionally the symptoms develop in the early stages of syringomyelia or as a result of tumor or hemorrhage of the spinal cord. Focal lesions in the spinal cord produce various symptoms, according to their situation.

The Cranial Nerves.—The olfactory, optic, oculomotor, pathetic, abducens, auditory, and glossopharyngeal nerves have been described in connection with the special senses. Destructive lesions of the *trigeminal nerve* of the motor portion causes paralysis of the pterygoid muscles. If they are unilateral, it is impossible for the patient to move the mouth toward the opposite side when the lower jaw is protruded.

FIG. 42



Cutaneous nerves of the head and face.

V_1 , V_2 , V_3 , first, second, and third branches of the trigeminal; SO, supra-orbital; l , lacrymal; st , supratrochlear; it , infratrochlear; e , ethmoidal; sm , malar; at , auriculo-temporal; b , buccinator; m , mental; am , auricularis magnus; oma and amc , occipitalis major and minor.

The soft palate is sometimes flattened on the affected side, and occasionally the uvula deviates to the sound side. Irritative lesions produce cramp known as trismus, usually due to central disease. The sensory portion of the trigeminal supplies the skin of the face and the mucous membranes of the cavities of the head. The distribution of the three branches is shown in Fig. 42. Irritative lesions produce tic douloureux; destructive lesions, anesthesia in the distribution of the part affected. The Gasserian ganglion, situated in the floor of the middle fossa of the skull, is the ganglion of the sensory portion of the nerve, and corresponds to a spinal ganglion. Irritative lesions may cause neuralgias, usually associated with disturbance of the various secretions, such as

tears, saliva, perspiration, or vasomotor phenomena. Destructive lesions (operative removal) produce, in addition to the areas of anesthesia, various trophic lesions, particularly ulceration of the cornea, or chronic ulcers in the mucous membrane of the mouth. There is usually diminished secretion of tears and of saliva, although occasionally a paralytic increase of the latter may occur. The facial nerve is the motor nerve for the muscles of the face. Unilateral destructive lesions produce paralysis of the muscles of the face (Bell's palsy). This can be recognized by disappearance of the folds, drooping of the corner of the mouth, and inability to close the eye. There may be loss of taste and hyperacusis in the ear on the same side. Occasionally there is deviation of the tongue, the palate is oblique, and the uvula is pulled toward the second side. Secretion of saliva on the same side is diminished or abolished. If the peripheral portion of the nerve is involved, usually both the upper and lower branches are affected, and the paralysis is general. If the lesion is intracranial, other cranial nerves, especially the auditory, are likely to be involved. The muscles give the characteristic reactions of degeneration. If the lesion is central, the upper branch commonly escapes, or, at least, instead of being paralyzed, is only paretic. Moreover, in central lesions lying above the pons there is also hemiplegia. In facial paralysis it is almost impossible for the patient to masticate on the diseased side, because food collects between the cheeks and the gums. It is also impossible for him to whistle. Saliva freely dribbles from the drooping corner of the mouth, and it is impossible to contract the orbicularis palpebrarum; the eye remains open even in sleep (lagophthalmos); the corneal reflex is abolished or imperfect. When the patient attempts to close the eye, the ball rolls upward and outward. In addition, the palatine reflex also disappears. Irritative lesions of the facial nerve cause spasm of the facial muscles, usually spoken of as facial tic. The vagus nerve supplies motor fibers to the larynx, sensory fibers to the lungs, and inhibitory fibers, probably sensory in nature, to the heart. This nerve also probably sends sensory fibers to the gastro-intestinal tract. Destructive lesions of the vagus produce, if unilateral, paralysis of one vocal cord, interference with deglutition, and transient tachycardia. The laryngeal changes are most characteristic. Irritative lesions produce spasm of the glottis, with dyspnea or aphonia. The spinal accessory nerve is the motor nerve for the trapezius and part of the sternocleidomastoid. Destructive lesions of this nerve are the chief cause of torticollis. The hypoglossal nerve is the motor nerve for the tongue, and is, therefore, concerned in chewing, swallowing, and speaking. Unilateral destructive lesions produce paralysis of one-half of the tongue, which is protruded toward the paralyzed side, with atrophy and degeneration of the muscle. Fibrillary twitchings are usually present. Bilateral paralysis produces severe symptoms. The tongue lies flaccid in the mouth; it is impossible to protrude it, or even to move it from side to side. Mastication is impossible, and swallowing exceedingly difficult. Speech is at first

seriously affected. Paralysis of the tongue as a result of central lesion almost never occurs.

Mental Disturbances.—Mental disturbances may be divided into disturbances of consciousness (see First Sight Impressions) and disturbances of intellect. The mildest form of *disturbance of intellect* consists in impairment of memory or *amnesia*. This may be restricted to the memory of certain things only, as the names of certain classes of objects or certain groups of words. It may also be restricted to loss of memory for certain definite periods of time, which may occur as a result of severe injury or disease during or about this period. If the memory is lost for the period preceding the traumatism the condition is spoken of as antero-active amnesia; if for the period following, retro-active amnesia. Memory is commonly impaired in old age, and often as a result of chronic cerebral disease, particularly in paralytic dementia. General impairment of the intellect is manifested in a great variety of ways. Congenital failure of development is spoken of as imbecility or idiocy. In its milder forms imbecility consists in diminution of the reasoning powers. In its severer grades, and particularly in the more pronounced forms of idiocy, intellectual activity may appear to be absolutely abolished. Both conditions are usually associated with alterations in the substance of the brain, either in the form of hydrocephalus or of the various sclerosis associated with epilepsy. General impairment of the intellectual powers is spoken of as dementia. In its most typical form this occurs among the aged: the subject has loss of memory for recent events, is confused and querulous, and the reasoning powers are defective. Dementia also occurs as a terminal stage in other forms of insanity, such as periodic insanity, Huntington's chorea, etc. Disturbance of the intellectual functions associated with excitement and more or less violence is usually spoken of as *delirium*. Among the commoner symptoms of intellectual disorder usually grouped under the term insanity are exaltation or *mania*, depression or *melancholia*, and delusional states or *paranoia*. By *mania* is meant excessive intellectual activity, characterized by a tendency to be noisy, to be active, fondness for singing, shouting, swearing, or punning. There are usually, also, in the acute forms, a rapid loss of weight and decrease in the physical powers, while the patient believes himself to be in the most admirable and exceptional condition. Mania occurs as a result of the inflammation of the brain substance in *acute delirium*. It occurs in the exacerbations of general paresis and in diseased states of unknown etiology that are known by the term itself. In *melancholia* the expression of the patient is mournful: he is commonly quiet, sits with his head bowed, refuses to speak, to eat, or to take any interest in what is said about him. Often he weeps or groans, and when persuaded to converse expresses an acute sense of his sins and the hopelessness of his condition, or will complain of misfortunes that have not befallen him. *Melancholia* occasionally occurs in general paresis, and is one of the commoner forms of insanity. The term *paranoia* is used by different authors

in very different senses. In general, it may be said that the majority imply by it the existence of delusions or false ideas that have, among themselves, a certain logical sequence, or, as the term is, are organized. Thus a paranoiac may believe that he is being persecuted by a certain person and be able to give reasons why his persecutor should torment him. It must not be forgotten that occasionally these delusions may be true in fact, although none the less symptoms of the mental condition. When there is merely a false idea it is spoken of as a *delusion*. If the person complains of certain physical impressions, such as non-existent sounds, visions, odors, or tastes, the term *hallucination* is generally employed.

CHAPTER XVIII

THE TEMPERATURE

THE average normal temperature of the body is 98.6° , but is subject to certain physiological variations. In the early hours of the morning it may be a degree or so lower than the evening, when it attains its maximum rise of the day (diurnal fluctuation). Violent exercise, mental exertion, eating, and exposure to overheated atmosphere raise the temperature. It is modified by age; in infants and young children the temperature has a somewhat higher range and is subject to greater variation than in later life; in the aged the temperature may be subnormal.

Mode of Determination of Temperature.—The temperature of the body may be roughly estimated by the sense of touch, but this method is open to many sources of error. To insure accuracy a clinical thermometer should be employed. The better ones are provided with an indelible index and the mercury remains at the highest level to which it rose when the thermometer was in the mouth or axilla. When the instrument is not provided with such an index, the reading must be made while the thermometer is still in position.

In this country the temperature, as a rule, is recorded in the Fahrenheit scale. In European countries the Centigrade scale is employed. To convert Centigrade to Fahrenheit when above zero, or *vice versa*, use the following formulæ: $9 \times C \div 5 + 32 = F$, and $F - 32 \times 5 \div 9 = C$. The mouth is the usual place to take the temperature. The thermometer should be placed under the tongue and held in place by firm pressure of the lips, not the teeth. The thermometer should in every case be left in the mouth from three to four minutes. The *axilla* may also be employed as a place to take the temperature. The skin is wiped dry and the thermometer then placed in position, the arm being brought gently across the chest and held there for five to seven minutes. The axillary temperature is about one-half degree lower than that of the mouth. In infants and delirious, unconscious, and dyspneic patients the temperature may be taken in the *rectum* or *vagina*. The rectum must first be emptied of scybalous masses. The thermometer is oiled before slipping it in and is left for two to four minutes. The temperature is found to be about one-half degree higher than that of the mouth. If great accuracy is required the rectum should always be selected as the place to take the temperature, for thus extraneous factors, as the taking of hot or cold drinks or breathing of cold air, are avoided. The surface temperature is taken by a special thermometer with a large,

flattened base and held in place by an elastic strap. It should be left in place at least ten minutes. The temperature of freshly passed urine corresponds exactly with that of the body and may be taken if malin-gering is suspected.

Frequency of Observation.—In afebrile cases the twice-a-day taking of the temperature is sufficient to give warning of possible febrile complications. In continued fevers the temperature should be taken every three to four hours. In fevers that show rapid and great change in the temperature it should be taken with greater frequency. The temperature should always be observed immediately after a chill and one hour later, as the febrile changes are very rapid after the rigor.

FEVER

This word is used as a synonym of pyrexia (elevation of temperature), but more correctly should be used also to indicate the constitutional changes that occur with heightened temperature.

Normally the temperature of the body depends upon the constant control of heat production (thermogenesis) and heat loss (thermolysis) by the heat-regulating mechanism (thermotaxis). Thermogenesis is mainly the result of oxidation processes taking place in the skeletal muscles and the glandular structures, particularly the liver. Thermolysis is chiefly through pulmonary expiration and evaporation of water from the skin. Thermotaxis is the control of temperature by the heat centres in the brain. In febrile conditions there is a disturbance of the heat-regulating mechanism, associated with increased heat production. It is not definitely settled what causes this disturbance in the heat-regulating mechanism, though certain conditions are known clinically to cause fever. Vaughn and others advance the theory that the bacterial fevers are a manifestation of anaphylaxis. His explanation briefly is as follows: the constantly maintained bacterial matter during an infection acts as a foreign protein which is split up (cleaved) by the body ferments. The reaction between these toxic products of cleavage and the tissues, as also the stimulation of cellular activity necessary to produce specific proteolytic enzymes to digest the foreign protein, cause fever in its broadest sense. In a similar manner, the so-called aseptic fevers, occurring after operations, are the result of toxic cleavage products of parenterally introduced (*i. e.*, not introduced through the alimentary tract) proteid as blood, fibrin, etc. The fever that occurs in certain cerebral lesions, *e. g.*, sunstroke and thrombosis, may be partially explained by the above and partially by the additional factor of the process directly involving the heat centres.

Causes of Fever.—It is thus seen that fever may be the result of (1) *abnormal bacterial invasion* of the body whether it be an acute infectious disease or a local inflammatory process; (2) *intoxication* through the introduction into the system of chemical poisons, exogenous

toxins which are usually elaborated by bacteria, or endogenous toxins, the result of faulty metabolism; (3) *disturbance of the heat centres* by direct or reflex causes.

The Height of Temperature.—Variation in the temperature below 95° and above 106° are occasionally seen, but are not compatible with life if prolonged for more than a short time. Certain terms have been applied to the various degrees of temperature, to indicate in a general way the degree of fever:

Below	{ 35.0° C. = 95.0° F. 36.0 C. = 96.8 F.	Very low or collapse temperature.
About	36.5 C. = 97.7 F.	Subnormal temperature.
Normal	37.0 C. = 98.6 F.	Normal temperature.
	{ 37.5 C. = 99.5 F. 38.0 C. = 100.4 F.	Slightly above normal or subfebrile temperature.
About	38.5 C. = 101.3 F.	
	{ 39.0 C. = 102.2 F. 39.5 C. = 103.1 F.	Moderately febrile temperature.
About		
	{ 40.0 C. = 104.0 F. 40.5 C. = 104.9 F.	Highly febrile temperature.
About		
Above	41.0 C. = 105.8 F.	Hyperpyretic temperature.

(From Finlayson.)

Types of Fever.—In general we speak of fever as mild or grave according to its intensity. Certain distinct types of fever, however, are recognized by their duration and by the amount of difference between the highest and lowest daily variation of the temperature range.

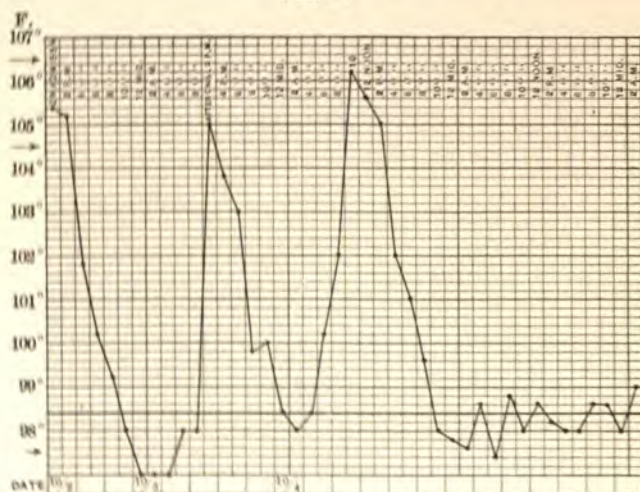
1. **Continued Fever.**—The daily variation is not more than 2° and the temperature continues for more than two days, falling abruptly, by *crisis*, or slowly, by *lysis*. Fever of this type is met with in lobar pneumonia, typhoid fever, typhus fever, erysipelas, and acute tuberculosis.

2. **Remittent Fever.**—The fever persists for more than two days, and there is a daily variation of more than 2°. This type of fever usually occurs sometimes in the course of any febrile disease, or in septicemia, pyemia, tuberculosis, in rapidly growing malignant growths, in local suppurations and inflammations, and estivo-autumnal malaria. The remissions usually occur in the morning, though they may take place any time of the day and are accompanied by sweating.

3. **Intermittent Fever.**—This type of fever is characterized by a periodical rise with fall of temperature to normal or below normal. When the paroxysms occur daily, the intermittent fever is quotidian in type; when every second day, one day intervening without fever, tertian type; every third day, two days without fever, quartan type. The representative types are seen in malaria but are simulated by a number of conditions, viz.: (1) relapsing fever and typhoid fever in rare cases; (2) suppuration without drainage (except brain abscess which causes subnormal temperature); (3) ulcerative endocarditis; (4) tubercu-

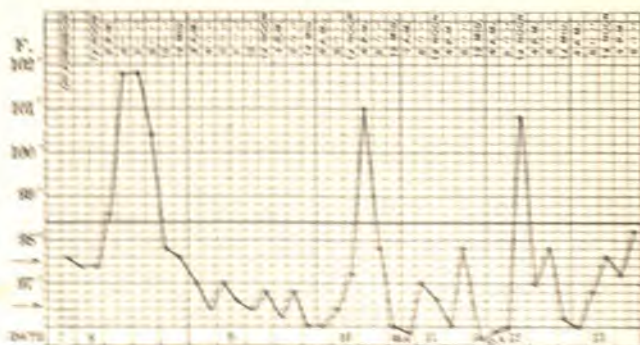
losis: (a) early stages of bone, pulmonary, or glandular tuberculosis; (b) pulmonary tuberculosis after the formation of a cavity; (5) occasionally Hodgkin's disease, leukemia, and anemia; (6) syphilis: the fever of the primary stage; (7) urinary intermittent fever: (a)

FIG. 43



Malarial intermittent fever, quotidian type. (Original.)

FIG. 44

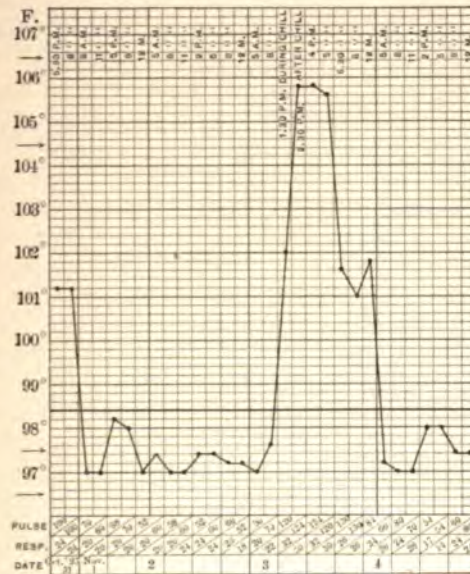


Malarial intermittent fever, tertian type. (Original.)

after passage of urethral catheter or sound and at times during passage of a calculus in ureter; (b) suppuration in genito-urinary tract; (8) hepatic intermittent fever: (a) gall-stones in or suppuration of bile passages; (b) external pressure on bile ducts causing obstruction; (c) acute inflammatory disease of liver; (9) prolonged use of morphine.

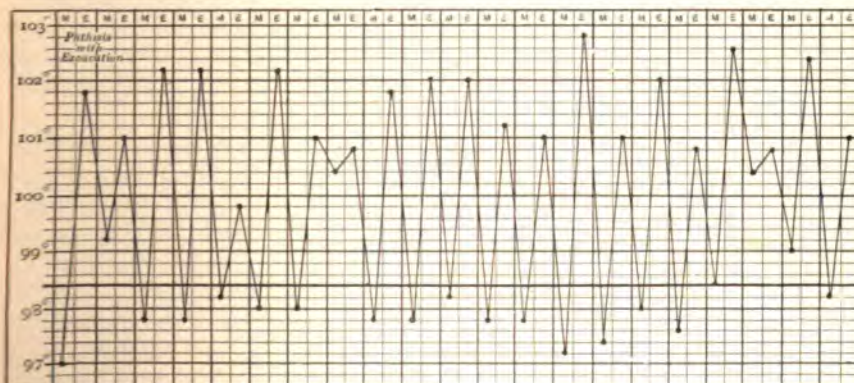
4. **Atypical Types.**—*Irregular fever* is characterized by marked irregularity in the range of the temperature, and by the absence of periodicity of the daily rises and falls, the highest or lowest point occurring at any time during the day.

FIG. 45



Malarial intermittent fever, quartan type. (Original.)

FIG. 46



Intermitting fever of tuberculosis. (Original.)

Recurring fever or recrudescence of fever is manifested by a return of fever after several afebrile days. This may be due to overexertion, excitement, insomnia, indigestion, constipation, and many other trifling

TEMPERATURE DIAGNOSIS

and some fevers have no effect upon temperature or it may be a remission (relapse).

The daily range of temperature in fever generally shows diurnal variations. At times, however, there may be nocturnal range, the exacerbation of the temperature occurring and the remission in the evening. Inversion is not infrequently in tuberculosis and in rare instances in other pulmonary diseases.

Types. It is important to remember that the three types are closely correlated and that the types frequently run into one another. This is particularly true in the continued type under the intermittent type of fever. In the intermittent type is not constant and may be continued type for a longer or shorter period, the temperature alternating from time to time.

Significance of the Temperature Curve.—Continued, fevers frequently follow a definite course, which may be divided into three stages: (1) the prodromal stage; (2) the initial stage; (3) the stage of decline.

The prodromal stage is often marked by malaise, headache, lassitude, and general discomfort. The initial stage or onset may be sudden or gradual. The prodromal stage may last only a few hours. It begins with a severe rigor or merely a subjective feeling of chill. The face of the body is pale or cyanotic, moist and clammy. The temperature in the mouth or rectum will be far above normal. At the beginning of the chill the skin becomes reddened. In febrile diseases, sudden in onset, are pneumonia, erysipelas, acute ulcerative endocarditis, acute gastritis or gastroenteritis, and acute infections, and acute gastritis or gastroenteritis. In children, especially if pain attends any inflammation, the rapidity and height of the rise of temperature is a good indication of the severity of the disease.

In the initial stage it may last for four or five days. A slight chill at the very beginning of the fever is a good indication of the severity of the disease. Typhoid fever characteristically shows a gradual rise, as do the majority of infectious diseases.

The stage of the pyrexia—the *fastigium*—the temperature rises to its highest point and remains almost stationary between maximum and minimum. It may last from two days to three or more weeks. The temperature usually returns to the maximum point of the first stage. The fastigium should only last a short time. The persistence of the fastigium indicates that the case is one of a greater severity or that there is a complication. It is

usually significant of the latter. In measles the complication is usually bronchopneumonia; in scarlatina, nephritis, inflammation of the serous membranes or middle ear disease; and in typhoid fever, a secondary infection, a relapse or the development of tuberculosis.

During the period of *defervescence* the temperature falls to normal. The termination may be sudden, by *crisis*, and accompanied by profuse sweating, increased flow of urine, and general alteration of the patient for the better. At times a *pseudocrisis* is seen, the temperature falls before the time of true crisis only to mount again rapidly and to remain up until the true crisis takes place. In other cases a gradual termination of the fever, *lysis*, occurs. The temperature falls a degree or two each day until the normal is reached. In this case sweating is less pronounced but may recur for several days. The slowing of the pulse and the general improvement likewise take place gradually. *Rapid lysis* is a term applied to a gradual but rapid fall of temperature. Diseases of sudden onset usually terminate by crisis, particularly lobar pneumonia, measles, erysipelas, malaria, chickenpox, and relapsing fever; if such diseases terminate by lysis a complication is indicated. The commonest examples of termination by lysis are typhoid fever, rheumatism, bronchopneumonia, scarlet fever, pleurisy, and septic infections. A sudden fall of temperature indicates the crisis unless the patient, instead of presenting the expected signs of improvement, presents the symptoms of grave illness. Such falls are the result of shock or collapse, or hemorrhage especially in typhoid fever.

Symptoms of Fever.—Pyrexia, a temperature over 99°, is not the only evidence of fever; in addition, certain metabolic changes occur as well as blood, circulatory, respiratory, gastro-intestinal, and cerebral disturbances.

The Blood.—Anemia is a constant accompaniment of fever, and in many of the febrile diseases there is a leukocytosis. Typhoid fever, malaria, uncomplicated tuberculosis, measles, and influenza are exceptions to this latter rule as they are accompanied by a leukopenia. In most of the acute infectious diseases there is frequently a concomitant bacteriemia.

Cardiovascular System.—Acceleration of the pulse is one of the constant phenomenon that attend pyrexia. As a rule the pulse-rate increases about eight beats for each degree Fahrenheit of elevation of the temperature above normal. This rule does not hold in certain infectious diseases, as typhoid fever, in those diseases causing pressure on the base of the brain, as basilar meningitis, and in febrile diseases complicated by a cardiac lesion in which there is a normal diminished pulse-rate, as coronary sclerosis. On the other hand, some diseases usually accompanied by fever, as diphtheria, peritonitis, and tuberculosis may run an afebrile course and yet the pulse-rate be much accelerated. The pulse-rate, moreover, gives indications of prognostic value: if the pulse is markedly increased it indicates myocardial degeneration and insufficiency, a danger signal to be closely followed.

In children, however, the pulse-rate, as a rule, is continuously rapid during a febrile course.

The arterial tension also shows alterations as the result of pyrexia. In the early stages of fever the pulse is large and hard, the arterial tension is high, and the vessels are full. In the latter stages arterial relaxation takes place and the pulse becomes small and feeble with low systolic pressure, probably as a result of cloudy swelling of the myocardium, and with increased pulse-pressure from relaxation of the vascular tonus.

Respiratory System.—The respirations are increased in fever, about two per minute to each degree of temperature.

Gastro-intestinal System.—There is anorexia, epigastric discomfort after meals, thirst, and nausea at times, the result of suppression of the secretions of the digestive glands, and constipation from the lack of intestinal fluids.

The Nervous System.—Hebetude, delirium, psychoses, and other nervous symptoms may attend fever. The severity and intensity of these symptoms depend not upon the height of the fever but rather upon the degree of toxemia.

If fever persists for a long time a low asthenic state may develop associated with stupor and sluggishness of the mental processes and with more or less continuous hallucinations of sight and sound. The stupor is attended with low muttering delirium and may be followed by complete unconsciousness, often with loss of power of the rectal and urinary sphincters. A converse condition may appear and is characterized by violent delirium, often severe headache, and, at times, especially in children, by convulsions.

Metabolism.—During fever the characteristic changes are those of increased katabolism. There is a marked breaking down of protein associated with a similar but somewhat less marked destruction of carbohydrates and fats and increased elimination of inorganic salts. This may be due in part to the action of the foreign proteins (*v. s.*) and partly to the fact that during fever food is usually withheld from the patient. The feeding of sufficient quantities of carbohydrates and fats during a febrile attack will usually prevent a marked increase in elimination of nitrogen.

There is a diminution in the amount of water excreted through the kidney due partially to increased elimination by respiration and to increased evaporation from the skin and partially also to retention of water from decreased functional activity of the kidney.

These abnormal metabolic processes cause two of the more pronounced symptoms of fever, viz., wasting, with loss of weight, and decrease in the daily output of urine which is high colored and of high specific gravity.

Resumé.—To recapitulate; fever causes:

1. Heightened temperature.
2. Increased pulse-rate.

3. Rapid respiration.
4. Thirst, anorexia, and epigastric discomfort and constipation.
5. Headache and hebetude, with, at times, coma and increased cerebation.
6. Wasting, loss of weight, and other evidences of disturbed metabolism.
7. Anemia.
8. Decrease in daily output of urine.

The Diagnostic Significance of Fever.—It has been shown that fever is caused by one of three conditions, viz.: (1) an infection; (2) a toxemia; (3) a central or peripheral irritation of heat centres.

There is always some cause for fever. If present, hysteria and malingering can usually be excluded. It indicates some morbid process occurring in the body. Usually the underlying condition is soon recognized after careful study of the physical signs and symptoms, but in a certain number of cases the recognition of the cause of the febrile process is not always possible. Therefore it is well to remember that hidden suppuration and pulmonary tuberculosis are the two conditions that are most likely to cause an apparent causeless rise in temperature. Occasionally the pyrexia may be the result of a mild or latent streptococcal infection or an atypical colon, typhoid, or paratyphoid infection.

Subnormal Temperature.—A subnormal temperature (hypothermia) may be physiological if it occurs in the aged or in those exposed to extreme cold. A pathological hypothermia results from a variety of causes. A temperature below the normal is a frequent and customary sequel of the diseases, with more or less prolonged pyrexia. It occurs in the course of wasting diseases, such as cancer, at times in anemia, and during starvation. It is seen habitually in myxedema, diabetes, scleroderma neonatorum, organic heart disease, alcoholism, tuberculous peritonitis in the latter stage, and in some forms of mental diseases as melancholia or in cerebral affections as brain abscess.

Shock, Collapse, and Hemorrhage.—These are important conditions causing a fall in temperature which is associated with a rapid, quick, small pulse, pallor, fall in blood pressure, relaxation of the sphincters, irregular shallow respiration, sweating, and marked prostration. *Shock* may be due to prolonged anesthesia, surgical operations, injury or trauma, intense pain, thrombosis or embolism of the brain, the rupture of an organ, particularly the stomach or intestines, and poisoning by powerful drugs or chemicals. If reaction occurs there is usually a rise of temperature to above normal.

Collapse is a term often used synonymously with shock, but strictly speaking it is the extreme depression and prostration that is, as a rule, followed by death.

Hemorrhages may be external or internal. The diagnosis of external hemorrhage is self-evident. Internal hemorrhage presents symptoms very similar to those of shock, in which, in fact, there is practically a

bleeding of the superficial, cerebral, and other arteries into the great vessels of the splanchnic region.

Localized Changes in Temperature.—Local variations in temperature from the normal are usually of minor importance. Increased heat over a part is a customary concomitant of a local inflammatory process. Localized coldness may be simply from exposure of the part to cold; otherwise it is a serious symptom as it indicates a failure of the circulation, usually in an extremity, and is the result of arteriosclerosis or thrombosis. It may be an early symptom of gangrene.

CHILLS

These vary from a passing creep or sensation of chilliness to a true rigor lasting a half-hour or even longer. In infectious diseases the milder forms are as significant as the more severe. The rigors are attended by general shaking, chattering teeth, pallid face, cold *skin* and *extremities* and cyanosis of the lips and finger tips. In spite of the extreme sensation of cold the internal temperature rises and may be 104° to 107° F. Clinically a chill or rigor marks the onset of severe infections which are initiated by a rapid rise in temperature. Recurrent chills at times accompany the rises in temperature that occur in those conditions which are characterized by intermittent fever (*v. s.*). Chills occurring in the course of continued fever usually indicate a secondary infection, or may be the result of internal administration of antipyretics.

True chills must be differentiated from the so-called nervous chills the result of excitement or fear and which are not accompanied by fever.

Shock, collapse, or hemorrhage may cause a subjective feeling of chilliness and coldness, but, again, there is an absence of fever, the temperature being subnormal.

SECTION IV

PHYSICAL DIAGNOSIS

CHAPTER XIX

GENERAL CONSIDERATIONS—METHODS

INSPECTION

By inspection we learn the size, shape, symmetry, color, and character of the movements of the whole or part of the body. The student should teach himself a method of observation by which all the facts are correlated in regular systematic order. He should accustom himself to always make the observations in a regular manner, noting first the size, then the shape, and then the other facts garnered by his inspection of the part or parts.

Methods of Inspection.—The whole body should be examined. The entire surface need not be exposed at once, but portions of the body can be covered and the various parts exposed sectionally. The light should be so arranged that it can be made to fall directly or obliquely on the surface. The patient must be seated or lying down in an easy, symmetrical position. He should be viewed by the observer standing first in front, then behind, and also from the side. To observe the anterior portion it is often well to stand behind the patient and look downward over the shoulders. The arms should fall by the side; the breathing should be quiet and undisturbed. In nervous diseases and diseases affecting the muscles and bones, the patient's gait, his ability to stand, and the method of rising or sitting should be observed.

Inspection of Cavities.—Inspection of many of the body cavities, as the nose, stomach, or rectum, is made possible by the use of special instruments. The methods of employment and the data obtained by the use of these is considered elsewhere.

PALPATION

By palpation is meant the employment of the sense of touch in the examination of accessible portions of the body. Palpation confirms and checks, in part, the results of inspection and obtains additional

information. We learn from palpation of the examined part: (1) the size, shape, and contour; (2) the degree of dryness, moisture or temperature of the overlying skin; (3) the sensibility, the absence or degree of tenderness; (4) the movability as determined by the palpating hand, or with respiration or functional movement, as gastric and intestinal peristalsis, and the character of the normal movements, as when a joint is examined; (5) the *resistance* and *density*, whether hard, soft, elastic, or edematous, and whether a swelling is solid, air-containing, or filled with fluid. The latter is ascertained by fluctuation or tremor of the fluid if the palpating hand is placed over the swelling and it is struck with a finger of the other hand.

In the palpation of special organs special phenomena are elicited which depend upon the function of that organ. Thus in palpation of the chest, the vibrations originating in the larynx during the act of speaking are transmitted through the pulmonary channels and tissues to the palpating hand—*fremitus*. Abnormal vibration from the friction of two roughened pleuræ can be felt, as can the intrapulmonary agitation of fluids (*friction rubs* and *rales* respectively). By palpation over the heart we determine the character and location of the point of maximum impulse of the cardiac contraction. Furthermore, abnormal vibrations originating in the heart or bloodvessels (*thrills*) can be recognized by the sense of touch.

PERCUSSION

By percussion is meant the act of striking upon any part of the body in order to throw the underlying structures into audible vibration and to judge, from the character of the sounds produced and the degree of resistance to the percussing finger, the physical condition of the parts beneath. Clinically percussion is employed to outline organs and to determine if there is more or less air than normal in the air-containing organs. For example, the quality of the sound produced over the heart changes when the margin of the organ is reached, and hence the cardiac area can be definitely outlined by noting the appearance of the change in sound. In the same way by percussing over the lung, an air-containing organ, changes in the quality of the normal sounds may indicate the presence of more air, *e. g.*, emphysema, or less air, *e. g.*, pneumonic solidification.

Method of Procedure.—Percussion may be performed with the fingers, with instruments, or with a combination of both. The blow may be struck upon the part directly (immediate percussion) or upon a medium interposed between the part percussed and the percussing finger or instrument (mediate percussion). The blow may be light (superficial percussion) or forcible (deep percussion). The sounds elicited may be listened to through a stethoscope placed upon the part percussed (auscultatory percussion) or attention may be directed

more to the feeling of resistance than to the character of the sound (palpatory percussion).

Technique.—The examiner should stand or sit directly in front of or behind the patient. The best position of the patient is standing with the muscles relaxed, the head straight and pointing forward and the arms hung loosely at the sides. Alterations in the position of the head or arms and contractions of the muscles causes changes in the percussion note, particularly noticeable when corresponding points of the lungs are compared.

While percussing the posterior thorax it is desirable to have the patient stoop slightly forward with the arms folded. If the patient is confined to bed, if not too ill, he should sit up during the percussion,

FIG. 47



Flint's plessor.

FIG. 48



Flint's pleximeter.

as contact with the bedclothes deadens the sounds elicited. If the body must be recumbent it should be as level as possible. If the posterior chest wall is to be percussed and the patient is turned upon his side it is of great importance to remember that there is a marked difference of the percussion note and other physical signs between the upper and lower sides of the chest.

Immediate or mediate percussion may be employed. If the latter, the striking finger or instrument is known as the *plessor* and the intervening medium, a finger of the opposite hand or a small disk of hard rubber, wood, metal, glass, or ivory, is called the *pleximeter*. The fingers are not only more convenient than instruments, but the sense of resistance can also be better appreciated. Only the finger which is used as a pleximeter must be placed over the surface to be examined in close and immovable contact without undue pressure, so that no intervening air can interfere with sounds elicited. For instance, if the anterior portion of the chest is to be percussed, the fingers must be placed between the ribs in the interspaces and not at right angles to them. The rest of the hand should rest lightly on the surface so that pressure from it will not interfere with the production of the sounds. Only one finger may be used; but if an organ is to be outlined, three fingers, with the palmar surface down, placed close together and laid flat upon the surface of the body and alternately percussed will often indicate a change in note more delicately and more exactly than if one finger is moved each time a note is struck. Several points in the use of the plessor

information, size, shape, and texture of the organ of tenderness; or with respiratory peristalsis, and joint is examined elastic, or edematous or filled with fluid if struck with a

In the palpation which depends on the force of the chest, the speaking are related to the palpation of two respiration of fluid over the heart, maximum impulsive vibrations are recognized by

By percussion in order to the to judge, from of resistance to beneath. Clinician determine if the organs. For the heart changes cardiac area of the change in an air-containing may indicate the *e. g.*, pneumonia.

Method of fingers, with the may be struck with a medium into the finger or instrument (superficial percussion) elicited may be part percussed

information, size, shape, and texture of the organ of tenderness; or with respiratory peristalsis, and joint is examined elastic, or edematous or filled with fluid if struck with a



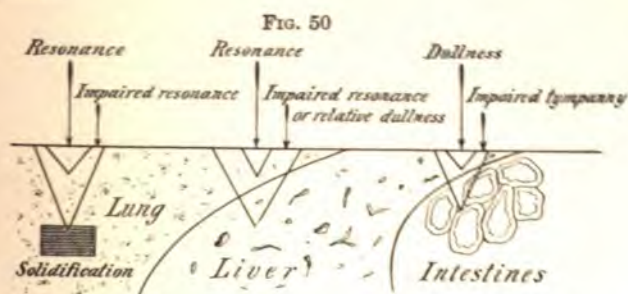
of the tissue thrown the force of the blow. In the tissue directly underneath is employed, to determine containing organs which may there is tenderness of the

situated parts into vibration It is used to determine the solid structures or to outline (solidifications) covered by air-

Immediate Percussion.—This form of percussion is useful to determine the degree of resistance rather than the actual percussion note. It is occasionally employed over the clavicle to determine the note of the underlying lung. It is of primary importance to compare only the notes over each clavicle and not to compare the clavicular note with that elicited over the tissue beneath as the clavicles are bony structures and are themselves capable of producing a change in note.

At times a smacking stroke with the flat of the hand is used to grossly estimate percussion changes over larger areas, as in large pleural effusions.

Immediate palpatory percussion may be performed by simply striking the organ with the ball of the finger and permitting it to remain there for a brief instant or so. The so-called finger-flicking (Wilson), the first or second finger being snapped off from the thumb on to the surface to be examined and lingering there for an instant, is employed to outline a superficial solid organ, as the spleen, overlying hollow structures, and the intestines.



Variations in sounds produced by light (small wedges) and deep (large wedges) percussion.

Bimanual palpatory percussion (Sewall) is performed by lightly resting the fingers of the palpatory hand upon the organ to be outlined and using the middle finger of the other hand as a plessor. When the striking finger reaches the organ, vibrations can be felt in it by the palpatory hand.

Mediate Percussion.—This is the method universally employed and the one that gives the best result to the inexperienced. It has already been sufficiently discussed. *Mediate palpatory percussion* is a form of mediate percussion in which emphasis is laid upon the sense of resistance rather than upon the elicited sound. To perform this the pleximeter finger of one hand is struck gently with the ball of the first three fingers of the other hand. The plessor finger lingers on the pleximeter finger a few seconds before repetition of the stroke. Palpatory percussion in any form is a valuable aid in outlining organs surrounded by hollow areas or in determining the levels of fluid in the abdominal or thoracic cavities, more particularly in observing the changes that occur as a result of postural variations, and in outlining contiguous structures of the same percussion note.

must be remembered. It consists in placing the bell of the middle finger of the right hand at right angles and kept in contact with the chest wall. The finger strikes the organ over which the strokes before moving the finger. The sound previously heard becomes abruptly fall directly and perpendicularly. This procedure is carried out in radiating pleximeter finger with a stroke. It is of value in outlining the position and rebounding like that of a ball. It is of value in outlining the position by movement of the wrist. It is of value in outlining the position of the finger alone may be used in defining the margins of limited areas.



—In this form of percussion the force is applied lightly. The middle finger, flexed at the proximal phalangeal joint, is used as a pleximeter, and is directed perpendicularly to the chest wall. The stroke is made upon the proximal end of the flexed portion of the finger. It is a particularly exact method of outlining the heart and great vessels of the thorax.

The Sounds Produced by Percussion.—

The sounds produced by percussion have a certain musical quality and may vary in pitch, volume, duration, and quality as musical sounds.

Pitch.—This depends upon the number of vibrations occurring in a definite time. It will be high when the vibrations are rapid, low when they are slow. In a solid or cavity contains the lower becomes

loudness depends upon the amplitude of the vibrations. It is proportional to the square of the amplitude. It is the loudness employed and essentially is the loudness

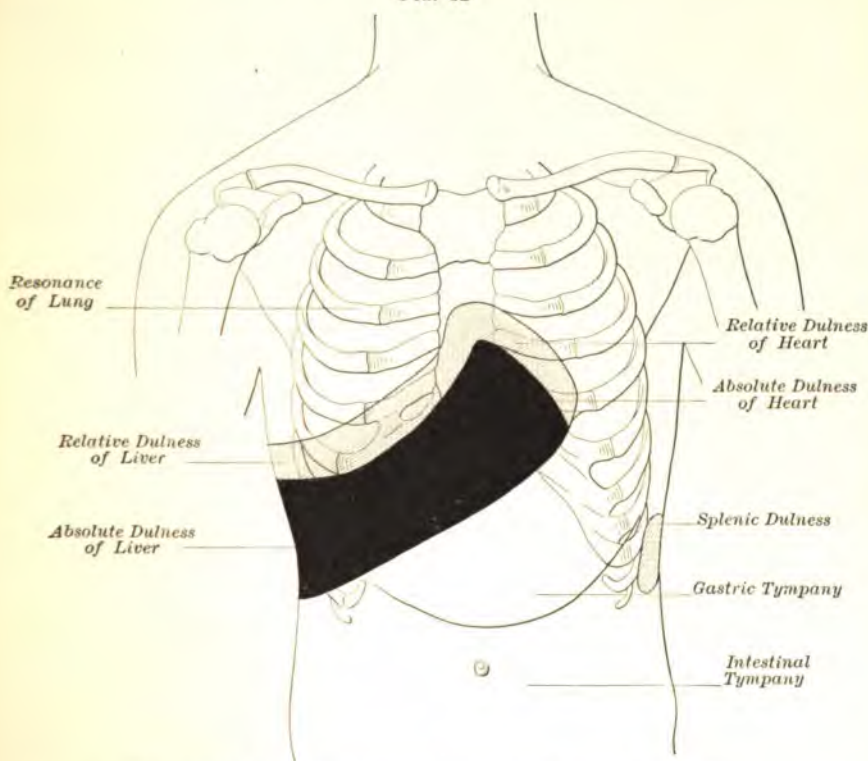
Force of sound.—The force of a sound depends upon the length and amplitude of the vibrations and varies directly with the pitch and duration. Sounds that are high in pitch are of short duration. The quality of a sound by which it is distinguished from other sounds is the quality. Changes in the pitch may change the quality.

Deep.—The sounds produced by percussion: (1) resonance or hyper-resonance, (2) dulness. Modifications of these types are produced under every variety of circumstance. Resonance is applied to the clear sound produced by percussion of the lungs. When the resonance is increased, the sound is known as *hyper-resonance*.

resonance, and results from an increase of intrapulmonary air. When there is a decrease or muffling of resonance it is known as *diminished* or *impaired resonance*, and results from small pulmonary solidifications or slight thickening of the pleura.

Dulness.—Dulness indicates the absence of air. *Absolute dulness* or *flatness* is a term applied to the percussive note over a solid organ. It is a high-pitched note of short duration.

FIG. 52



Showing the areas of resonance, dulness, and tympany in health.

Relative dulness indicates contiguity of air-containing structures to a solid organ. The note is low pitched and of greater duration than that of absolute flatness. The degree of dulness may also be expressed by the terms slight, moderate, or marked dulness.

Tympany.—This is the term applied to the sound that is produced over an air-containing cavity with smooth walls. It is a low-pitched note of large volume and marked duration, possessing a peculiar metallic quality which is best described as hollow. As tympany approaches and is modified by dulness it is termed a moderate, dull, or slight tympanitic note.

Special forms of tympany are: *cracked-pot resonance* and *amphoric resonance*. The former sound is readily imitated by sharply striking upon the knee, the hands so clasped that there is a slight amount of air between them. It is produced by a quick stroke over a pulmonary cavity or a pneumothorax communicating with a large bronchus, when the patient's mouth is wide open, and is best heard by placing the bell of the stethoscope in front of the open mouth. It may also be elicited by percussion of an infant's chest during crying or by forcible percussion of the lung of an adult near the trachea, provided he has a yielding thorax. The latter sound possesses the characteristic of the sound produced by striking a large jar with a wide mouth and is produced by percussing over a large smooth-walled cavity with a small opening.

The Degree of Resistance.—This is estimated by the sense of touch. When air-containing structures are percussed the sensation to the fingers is as if the part underneath bounded away. Over solid structures the sensation is as if the tissues beneath were immovable. The sense of resistance is best appreciated by immediate or by palpatory percussion.

AUSCULTATION

Auscultation is the method of physical examination employed to recognize the body sounds and to interpret variations in them from the normal.

Methods of Auscultation.—The patient should lie, sit, or stand in an easy and unrestrained position. Two methods may be employed in auscultation: the immediate or direct, and the mediate or indirect. The former method is of value only in the auscultation of the chest or abdomen, the ear being placed directly upon the skin with only a thin towel intervening. The gross characteristic sounds are easily elicited by this method, but for finer and more careful work, particularly in the auscultation of the heart with the necessity of localizing and timing sounds, the mediate method by means of the stethoscope, except in appreciating deep-seated pulmonary lesions, is of greater value.

The Stethoscope.—The stethoscope consists of a bell which is placed upon the auscultated organ and of one or two ear-pieces which are connected by flexible or rigid tubes. The single stethoscope has but one ear-piece, the tube is usually firm, and is of particular value in recognising the shock of an aneurism or impulse of the heart. The disadvantages of this instrument are that the weight of the head at times causes a pressure sufficient to cause pain, if the chest is sore, or to modify the sounds in auscultation of bloodvessels or structures in close proximity to the ear and that the listener is confused by extraneous sounds.

The *double or binaural stethoscope* has two ear-pieces. The advantages of this kind of a stethoscope are: it can be applied lightly over a

tender part; the listener is not obliged to stoop or assume positions in which the blood is forced to his head, with a consequent ringing in the ears; outside noises are largely obviated by the well-fitting ear-pieces and the pressure over a part, especially the heart, is easily regulated. The bell of the stethoscope is placed lightly but closely to the bared part so as to exclude outside noises. It is held in place by the fingers and thumb, care being observed not to have the fingers touching the patient. *The differential double stethoscope* has two bells, the sounds from each being conveyed to a different ear. *The phonendoscope* is a flat, round, shallow, metallic bell covered by a vibrating hard rubber disk and connected by two soft rubber tubes. It is used to intensify sound.

The Bowles stethoscope is constructed on a principle similar to that of the phonendoscope, but a single rigid tube is attached to it at right angles to the central axis. The tube may be slipped into the bell of an ordinary binaural stethoscope.

The multiple stethoscope for the use of classes has several sets of ear-pieces, attached to one chest-piece by means of branched tubing.

Errors of Auscultation.—In auscultating it is necessary to disregard external noises, noises produced in the stethoscope and

FIG. 53

Hawksley's single stethoscope.
(Two pieces.)

FIG. 54



Binaural stethoscope.

muscle sounds, and to avoid producing sounds by friction of the stethoscope upon the skin or of the fingers upon the stethoscope. The bell must be exactly approximated to the skin and the tubes kept from becoming blocked, kinked, or disconnected. If the ear-pieces do not properly fit the ears or are inserted upside down, outside noises are likely to obscure the body sounds.

Graphic Record of Physical Signs.—The employment of graphic signs to represent physical findings is of practical value to the beginner, as only by using them or by actually writing down the various physical

signs as elicited is he able to properly coördinate his findings and from these arrive at a definite conclusion.

Graphic signs also afford an easy and accurate method of preserving records for future use. Numerous symbols have been devised to represent graphically the various physical signs, but in the end every man using these aids will probably modify the symbols of others or employ some of his own devising. Therefore the symbols used in the following list, many of them borrowed from Wyllie of Edinburgh and Sahli of Bern, are simply offered as suggestions and are employed in the plates to simplify the explanations and descriptions.









Explanation of the Symbols Used in the Plates Illustrating Special Diseases

Percussion-sounds.—Blue shading = Areas of superficial dulness; the intensity of the color expresses the intensity of the dulness.

HR = Hyperresonance.

T = Tympany; the pitch is indicated by a dot above or below the letter.

Breath-sounds.—An ascending line indicates inspiration; a descending line, expiration. The length of the line shows the length of the sound; the thickness, its intensity. A dot above or below the line indicates high or low pitch. Two cross lines are used to designate bronchial breathing; a single cross line indicates bronchovesicular breathing. An interrupted line stands for cog-wheel or interrupted breath-sounds.

- | | |
|---|---|
|  | = Normal vesicular breath-sounds. |
|  | = Weak vesicular breath-sounds. |
|  | = Harsh vesicular breath-sounds (puerile breathing). |
|  | = Harsh vesicular inspiration, prolonged vesicular expiration. |
|  | = Sharp vesicular inspiration, slightly prolonged vesicular expiration. |
|  | = Interrupted (cog-wheel) breath-sounds. |
|  | = Bronchial breath-sounds (bronchial breathing). |
|  | = Bronchovesicular inspiration, low-pitched prolonged expiration. |

Rales.—Sonorous and sibilant rales are represented by undulating lines, the length corresponding to the duration, while a dot above or below the line indicates the pitch.

crepitant, and crepitant rales are represented by a circle of which indicates the size of the rales. An arrow drawn through the circle shows that the rale is heard during inspiration, a descending line that it is heard during expiration. Mucus rales heard over consolidated areas—rales sonantes—are indicated by large or small dots, according to the size of the area.

- = Sonorous rales.
- = Sibilant rales.
- = Crepitant rales.
- = Small bubbling (subcrepitant) rales.
- = Large bubbling rales heard during both inspiration and expiration.
- = Large and small bubbling rales.
- = Crepitant rales heard over solidifications.
- = Subcrepitant rales heard over solidifications.
- = Large bubbling rales heard over solidifications.
- = Large and small bubbling rales heard over solidifications.
- = Crepitant rales, to be heard only during inspiration.





Friction-rub.

- = Friction-rub, as heard over any serous surface.

Heart-sounds.—The symbols used to indicate the feet in Latin poetry are used to represent the heart-sounds. The straight line indicates the first or systolic, the curved lines the second or diastolic sound. The length of the line shows the relative as well as the absolute loudness. The length of the line and the extent of the curve indicate the length of the sound.

- = Normal heart-sounds.
- = Long loud first sound.
- = Normal first sound, accentuated second sound.
- = Loud first sound, reduplicated second sound.
- = Reduplicated first sound, accentuated second sound.

Murmurs are represented by short parallel lines either horizontal or vertical, varying in length according as the murmur increases or decreases in intensity. The thickness of the lines shows the loudness of the murmur, the number of lines shows its duration.

-  = A soft murmur, commencing distinctly and gradually fading away.
 = A loud murmur of the same character.
 = A short loud murmur, increasing in intensity (type of presystolic murmur).
 = Loud first sound, slightly accentuated second sound; short loud presystolic murmur, increasing in intensity to end with the first sound; long, soft, systolic murmur.

Fremitus.

- F +** = Increased fremitus.
F - = Diminished fremitus.
NoF = Absent fremitus.

Other Symbols.

- X** = Impulse.
M = Margin (of an organ).
R = Retraction.
B = Bulging.
v = Visible.
p = Palpable.
Xvp = Visible and palpable impulse.
Mvp = Visible and palpable margin.

CHAPTER XX

PHYSICAL DIAGNOSIS OF DISEASES OF THE HEART AND BLOODVESSELS

Topographical Anatomy.—Outline of Heart on Chest Wall.—(See Plate IV.) To have a general idea of the form and position of the heart, map its outline on the wall of the chest as follows:

(a) To define the base—*i. e.*, the part to which its great vessels are attached—draw a transverse line across the sternum corresponding with the upper borders of the third costal cartilages; continue the line 1 cm. to the right of the sternum and 2.5 cm. to the left.

(b) To find the apex, mark a point 2.5 cm. to the sternal side of the midclavicular line, between the fifth and sixth ribs.

(c) To find the lower border (which lies on the central tendon of the diaphragm) draw a line, slightly curved downward, from the apex across the bottom of the sternum (not the ensiform cartilage) as far as its right edge.

(d) To define the right border (formed by the right auricle) continue the last line upward with an outward curve, so as to join the right end to the base.

(e) To define the left border (formed by the left ventricle) draw a line curving to the left, but not including the nipple, from the left end of the base to the apex.

Such an outline shows that the apex of the heart points downward and toward the left, the base a little upward and toward the right; the greater part of it lies in the left half of the chest, and that the only part which lies to the right of the sternum is the right auricle. A needle introduced in the third, fourth, or fifth right intercostal space close to the sternum would penetrate the lung and the right auricle. A needle passed through the first intercostal space close to the right side of the sternum would pass through the lung and enter the superior vena cava above the pericardium.

Valves of the Heart.—The *aortic* valve lies behind the third intercostal space, close to the left side of the sternum. The *pulmonary* valve lies in front of the aortic, behind the junction of the third costal cartilage with the sternum, on the left side. The *tricuspid* valve lies behind the middle of the sternum, about the level of the fourth costal cartilage. The *mitral* valve (the deepest of all) lies behind the third intercostal space, about 2 cm. to the left of the sternum. Thus these valves are so situated that the mouth of an ordinary sized stethoscope will cover a portion of them all if placed over the sternal end of the third

intercostal space on the left side. All are covered by a thin layer of lung, therefore we hear their action better when the breathing is suspended for a moment.

Physiology.—Properties of the Heart Muscle.—The heart muscle has five physiological properties: (1) stimulus formation; (2) excitability; (3) contractility; (4) conductivity; (5) tonicity. The power of stimulus formation and of conductivity seems to be inherent in the heart and peculiar to its musculature, while the other properties are common to all muscles. The property of stimulus formation is explained by the theory that the fibers of the myocardium produces a stimulus substance which, when a sufficient quantity, under sufficient tension, in the presence of certain inorganic salts, has accumulated, causes contractions of the cardiac musculature. The response to a stimulus, no matter of what strength, is followed by contraction of the heart to its fullest extent, and is succeeded by a period in which the stimulus material having been used up, the heart is unable to contract to any stimulation, weak or strong. The property of conductivity is that function by which the contraction waves are passed downward to cause complete contraction of the whole organ. The contraction of the heart normally starts at the most excitable portion, the sino-auricular node of Keith and Flack (the so-called "pacemaker of the heart"), located at the junction of the superior vena cava and the right auricle. From here the contraction passes downward through the auricular fibers to a specialized structure, the auriculoventricular node of Tawara, which is the commencement of the bundle of His proper, the muscle bundle which connects the auricle with the ventricle. Through this bundle of muscle or neuromuscular tissue, which divides and runs to the right and left ventricle to eventually ramify with the Purkinje fibers of the ventricular wall, communicating directly with the ventricular muscle fibers, the contraction passes and ventricular systole takes place, occurring about $\frac{1}{10}$ of a second after auricular systole. This myogenic theory of cardiac activity makes the nervous control of the heart secondary to the muscle control. The nervous influence in the heart is supposed to be a controlling one solely, the vagus having an inhibiting effect, the sympathetics having an accelerating effect upon the heart rate.

Action of the Heart.—The heart beats—that is, alternately contracts and dilates or relaxes—65 to 85 times per minute in an adult. In females, the frequency varies from 75 to 85; in males from 65 to 75. With each beat, blood is propelled through the vascular channels of the body and drawn from them to the heart chamber. The first effect is produced by the contraction of the heart, or the *systole*; the second by the relaxation, or *diastole*. Other events, as the act of respiration, contribute to the completion of the outflow and inflow of blood, particularly to the latter.

The completion of the act of contraction and of the act of dilatation make up one revolution of cardiac action, or, as it is termed¹

Events of the Cardiac Cycle.—The following events make up the cardiac cycle. During the ventricular systole (1) the ventricles contract; (2) the auriculoventricular valves close; (3) the blood is propelled from the ventricles into the arteries, the columns of blood in the aorta and pulmonary artery receive a shock from the impact of the new volume of blood, and their bulk increases. The movement of the blood wave from this cause and from the secondary contraction of the large vascular trunks produces pulsation of the peripheral arteries, the pulse. The contraction is immediately followed by *relaxation*—the *diastole*. (1) The blood columns in the aorta and in the pulmonary artery fall back upon the valves guarding their outlets, the aortic and pulmonary valves, and lead to their closure. At the same time (2) the auricles are filled by blood pouring in from the veins. (3) The auricular muscles contract upon the blood in the chambers, driving it into the ventricles (auricular systole).

Inspection. — The Heart.—*Method of Examination.*—The patient should be stripped, and good light should fall directly as well as obliquely on the surface. The patient, for accuracy, should be examined both in the upright and in the recumbent posture. The examination should not be confined to the heart and vessels. In the examination of a case of suspected heart disease, observation is made of the general and of the local color, as of the lips, the fingers, and the conjunctivæ, to determine the presence of cyanosis, pallor, or jaundice; of the feet, to discover dropsy; the face, to note the appearance of the countenance; of the neck, to note the state of the vessels—the veins as well as the arteries; the eyes, to note their prominence and any retinal changes; the thorax, to ascertain the presence of dyspnea.

The Precordia.—The precordia is the region of the chest which overlies the heart. In the study of the appearance of the precordia we observe (1) the degree of prominence or swelling; (2) the impulse and other pulsations; (3) the interspaces; (4) the color of the surface.

PROMINENCE.—The precordia may be unduly *prominent* in children who have had rickets and possibly some cardiac hypertrophy in childhood, the prominence persisting in later life. Swelling also occurs in hypertrophy or dilatation of the heart, in pericardial effusions, localized pleural effusions and pointing empyema, and in aneurisms in the region of the heart. In pericardial effusion the ribs and the interspaces alike project. The latter are full or even with the surface. The precordia may be *sunken*. Old pericarditis, but more frequently old empyema, causes sinking in of the region. It may be the result of rickets or of spinal curvature, and occurs in the condition known as "funnel-chest."

THE CARDIAC IMPULSE.—With each contraction of the heart there is a slight visible and palpable movement in the fifth space just above the midclavicular line. In children the impulse may be in the fourth space and in old people it may be depressed. The localized impulse by the heart pushing against the heart wall is frequently

alluded to as the "apex-beat." This, however, is not a movement produced at the apex of the heart, which lies about 2 cm. below and to the left, but is produced during the systolic erection of the heart upon the great vessels, the impulse being produced by the systolic movements of the right ventricle which normally lies continuously against the chest wall. It is the point of maximum impulse of the heart against the chest wall. In addition to this localized impulse a diffuse movement of the entire precordia may at times be made out when the heart is acting violently or when the chest wall is thin. At times the impulse of the heart can neither be felt nor seen in normal individuals, particularly those with thick, muscular, or fat chest walls.

The Position of the Cardiac Impulse.—Changes of Position in Health.—The point of maximum impulse is not a fixed point in health as the heart moves with the movements of the body, and hence, when the trunk is inclined to the left, it falls toward the left axilla as far outward as the midclavicular line or even beyond that point. It moves toward the right and downward in full inspiration, or may disappear entirely toward the completion of that act.

Change of Position in Disease.—The impulse may be displaced to the right or left, and upward or downward. These changes are due either to (1) *disease outside of the pericardium*, to (2) *disease within the pericardium*, or to (3) *disease of the heart itself*.

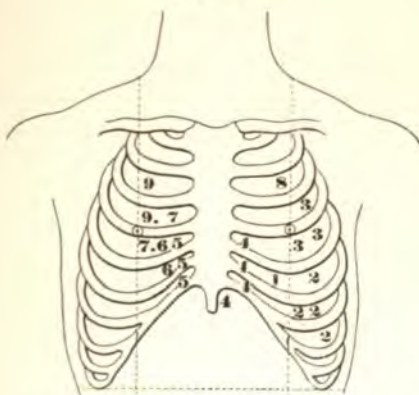
1. *Displacement of the Left.*—This occurs from (a) *alterations outside of the pericardium*. The heart and consequently the impulse may be pushed to the left by a right-sided pleural effusion or pneumothorax or a compensatory emphysema of the right lung, or pulled to the left by the contraction of adhesions between the pleura and pericardium. Displacement to the left and upward at times is seen in fibroid phthisis of the left upper lobe of the lung. In disease of the mediastinum (abscess, aneurism, or enlarged glands) the heart is pushed downward and toward the left. In disease of the abdomen the impulse is displaced. If the liver and spleen are enlarged, or if the abdomen is distended by ascites or meteorism, the diaphragm is raised, and, therefore, also the heart. The impulse is then seen to the left of the normal position, and may be one or two interspaces higher than normal. Dilatation of the stomach, from flatulence or disease, is a frequent cause of displacement of the impulse.

(b) *Alterations within the Pericardium.*—In cases of pericardial effusion the impulse is shifted to the left and upward. It is seen in the fourth and even as high as the third or second interspace. In this latter position the movement is the impulse of the right auricle and conus arteriosus against the chest wall.

(c) *Diseases of the Heart.*—The apex-beat is displaced to the left in dilatation and hypertrophy of the heart. In the latter it is also displaced downward. It may be as low as the sixth or seventh interspace and extend as far as the left as the anterior axillary or the midaxillary line.

2. *Displacement to the Right.*—(a) *Alterations outside of the pericardium.* The heart is pushed to the right in left pleural effusion or pneumothorax and in emphysema of the left lung. We find, moreover, in pleural contractions and fibroids phthisis of the right lung the heart drawn to that side. Under these circumstances the impulse is noted either in the epigastric region along the margin of the ribs or even to the right nipple line, or in any interspace from the third to the sixth, along the right edge of the sternum. (b) The impulse is not displaced to the right in alterations within the pericardium, or (c) in disease of the heart.

FIG. 55



Normal and abnormal impulses. 1, normal position of impulse; 2, various points of displacement to left and downward; 3, various points of displacement to left and upward; 4, impulse from enlarged right ventricle; 5, displacement to right; 6, dilated right ventricle; 7, displacement in fibroid phthisis; 8, impulse of conus arteriosus; 9, fibroid phthisis, right lung (right border of heart).

The Extent of the Cardiac Impulse.—In health the impulse is limited to an area from 1.5 to 2.5 cm. in diameter. The area may be increased when the individual leans forward, at the end of expiration, and during states of bodily or emotional excitement. It is more evident when the chest walls are thin, and less when they are thick.

In disease the area of impulse may be increased. The causes are: (a) *Disease outside of the pericardium.* The area is increased in chronic phthisis with fibrous adhesions, and in pleural adhesions when the lung is drawn away from the surface of the heart. It is increased when the heart is pushed against the chest wall as in aneurism or in diseases of the mediastinum. (b) *Disease of the pericardium* tends to increase the area of impulse if moderate effusion is present. It is also increased in the presence of pericardial adhesions. (c) *Diseases of the Heart.* The heart may be either hypertrophied or dilated causing an enlarged and diffuse impulse at times.

New Impulses.—New areas of impulse, the heart not being dislocated, e from enlargement of one of the cardiac chambers or from disease

of the bloodvessels. A new area of impulse in the second or third left interspace may be caused by pulsation of the conus arteriosus or the pulmonary artery in thin people; it may be due to exaggerated pulsation of the left auricle such as occurs in mitral stenosis; it may be occasioned by retraction of the lung in this region; it may be due to hypertrophy and dilatation of the right ventricle; or it may be due to aneurism of the aorta. A dilated right auricle may give rise to a new impulse to the right of the sternum, in the fourth or fifth interspace. A systolic impulse in the second right interspace may be seen in aortic insufficiency. Most new impulses to the right of the sternum, however, are due to dilatation or aneurism of the aorta or of the innominate artery, and they are usually present in the second or third interspace.

RETRACTION OF THE INTERSPACES.—A very slight retraction occurs normally with each cardiac impulse, because of the smaller size of the ventricles during systole and consequent pushing in of the interspaces by atmospheric pressure. The interspaces may be retracted from pericardial adhesions; the retraction may be limited to the apex or may occur in each interspace over the precordial region; it may occur with the systole or with the diastole. Pronounced systolic retraction commonly occurs in marked hypertrophy of the heart and is usually of the interspaces over and in the region of the apex (left axilla). Systolic retraction in the second (rare), third, fourth, and fifth space occurs when the right ventricle is markedly hypertrophied. This may occur synchronously with the cardiac impulse, and give a wavy appearance to the lower precordium with each heart-beat. On inspection behind a systolic retraction of the region corresponding to the left eleventh and twelfth interspaces is seen in adherent pericarditis. This is known as *Broadbent's sign*.

The Arteries.—By inspection we may be able to determine pulsation or any undue swelling or other change in the course of the vessels. With the exception of pulsation in the carotids, which may temporarily increase under excitement, pulsation of the vessels is not usually seen in health. In old people we can see the pulsation of the aorta (rarely) at the episternal notch, and often pulsations of the superficial vessels.

The Arteries in the Neck.—Temporary pulsation of the carotid arteries from excitement has been mentioned. Persistent pulsation is commonly seen in anemia and is quite marked in exophthalmic goitre. It is striking in aortic regurgitation. It often attends the vascular changes of old age, and may be due to a simple cardiac hypertrophy, aneurism, or atheroma. It is common in conditions of low vascular tension. The innominate artery, as well as the carotids, often pulsates visibly in the neck, and may be so large as to simulate aneurism. The subclavians may pulsate for the same reason; they may also be seen to pulsate if the lungs are solidified or shrunk by disease.

The Thoracic Aorta.—An impulse of the thoracic aorta is usually from aneurism. The pulsation is not always due to disease; the aorta may be pushed against the chest wall, or the lung structure which

overlaps it normally may be withdrawn. It must not be confounded with pulsations of the thoracic wall due to pulsating pleural effusion or to those diffused pulsations occurring in anemia and exophthalmic goitre.

The Abdominal Aorta.—The pulsation is usually seen in the epigastrium and does not usually mean that the vessel is diseased. It occurs reflexly in patients with dyspepsia or organic diseases in the upper abdominal tract. The shock of the pulsation is transmitted to the hand with considerable force. The impulse is diffused, but not expansile. *Epigastric* pulsation also may be due to transmission of the impulse of the aorta by enlargement of the pancreas or tumors of the stomach or the omentum. The transmitted pulsation is distinct. The impulse is a transmitted one when the tumor can be defined and when a sensation of lifting is transmitted to the hand. The physical signs of aneurism are absent. If the patient lies on the abdomen or in the knee-chest position, the tumor falls away from the aorta and the impulse is not readily transmitted. Epigastric pulsation is also caused by aneurism of the abdominal aorta.

FIG. 56



Studying pulsations.

While functional epigastric pulsation usually occurs in neurotic subjects, and hence in the earlier periods of life, yet such pulsation is frequently seen at the climacteric and in old age. Late in life, with such impulse, fibrous thickening about the pylorus or contraction of the omentum may easily be confounded with malignant disease. Fecal accumulations in the colon may be made to heave by the beat of the aorta and cause exaggerated epigastric impulse. The bowels must be emptied before definite conclusions are drawn.

An epigastric impulse due to one of the above-mentioned causes must not be confounded with the shock of an hypertrophied or overacting heart transmitted to the left lobe of the liver. In dislocation of the heart from disease within the chest the impulse may be seen to the right or left of the xiphoid cartilage. A systolic retraction of the epigastrium is usually due to a dilated hypertrophied right ventricle.

Smaller Arteries.—In endarteritis examination of the femoral, al, tibial, brachial, radial, and temporal arteries reveals dilated,

tortuous, often pulsating vessels. Elongation of the artery is seen, so that instead of a straight tube it becomes a sinuous canal turning and twisting at short intervals. In addition, in hypertrophy of the left ventricle arterial pulsation is a prominent sign, although more marked in the vessels near the heart, as the carotids. In regurgitation at the aortic orifice pulsation is also frequently seen.

Capillary Pulse.—The capillary pulse is seen under the finger nails or in the skin after hyperemia is induced by firmly stroking the skin with the nail. It may be seen inside the lips if a piece of glass is pressed against them. There is a rhythmical pulsation of the capillaries causing alternate flushing and pallor of the surface. When marked, it is significant of aortic insufficiency. An indistinct capillary pulsation may sometimes be observed in apparently healthy persons, and is not rare in fever states and other conditions of low vascular tension.

The Veins.—By inspection we note the presence of:

A. ENLARGEMENT OF THE VEINS.—The change in size may be general or local. In both instances there is interference with the venous return of blood.

1. *General Enlargement.*—General enlargement may be observed in all the veins, but is more readily studied in the jugular veins of the neck. Associated with the enlargement, general venous engorgement is observed. General enlargement is due to dilatation of the right auricle and ventricle, which in turn may have arisen from cardiac disease, or, on account of increased pulmonic blood pressure, from emphysema and other pulmonary obstructions. In rare instances pressure upon the cavæ by a mediastinal tumor may cause general overfulness of the veins.

2. *Local Enlargements.*—Local increase in fulness of the veins is due to narrowing or closure of the venous trunk by pressure or by thrombosis. A mediastinal tumor pressing upon the cava will cause abnormal fulness of the *jugulars*. The veins of the scalp become distended and tortuous in thrombosis of the longitudinal sinus. Enlargement of the veins of the *arm or leg* points to compression or thrombosis of the axillary or femoral vein respectively. Enlargement of the superficial veins of the *thorax* is seen in intrathoracic pressure from tumor or aneurism, rarely in dilatation of the heart. Enlargement of the veins of both *legs* may be due to obstruction of the vena cava or both iliac veins. The latter is liable to occur in pelvic tumors. When there is engorgement of the portal vein, collateral circulation is frequently carried on through the *abdominal veins*. The veins are enlarged and in some instances the veins about the navel become enormously distended because of a permanently patulous umbilical vein. The crown of veins—*caput Medusæ*—is significant of cirrhosis of the liver and of pyelothrombosis. Enlargement of the veins of the extremities from causes above mentioned must not be confounded with the unilateral or bilateral varicosity that occurs during and after pregnancy, after prolonged intra-abdominal pressure from other causes, and in inflammations of the veins in the course of septic diseases, as typhoid fever.

B. Pulsation of the Veins.—The circulation in the veins differs from that in the arteries. The blood flow is continuous. Two circumstances modify it—respiratory movements and cardiac action.

Pulsation Due to Respiratory Movements.—The modifications is particularly seen in the veins of the neck. During inspiration all of the veins empty rapidly, while in forced respiration, or with strong effort, as seen in coughing, the discharge from the veins is checked and they become full and even overdistended. When the fulness of the veins is normal the respiratory alternations are not observed, except the swelling that occurs in severe coughing, as in whooping cough. When they are abnormal, as from right-sided cardiac dilatation (*q. v.*), they show a corresponding to-and-fro swelling synchronous with respiratory movements. Upon coughing the jugular bulb may appear as a rounded pulsating bunch between the heads of the sternomastoid muscle. Increased pulsation with fulness of the veins is seen during the labored expiration of asthma and emphysema.

Alternation of the respiratory movements of the veins is observed in cases of pericarditis or of mediastinopericarditis. Normally the vessels are drawn upon and bent during the act of inspiration—inspiratory collapse. In the above-mentioned pathological conditions they swell in inspiration and empty during expiration, directly opposite to the normal state.

Pulsation Due to Cardiac Movements.—*The Venous Pulse.*—The cardiac movements also modify the movements of the blood in the veins. They cause rhythmical pulsation, or the venous pulse. (See graphic records of the pulse—the venous pulse).

Diastolic venous collapse is seen in pericarditis. The collapse occurs at the time of the cardiac diastole. It is distinguished from the true pulse as follows: compress the jugular vein—pulsation ceases above and below the seat of compression.

Palpation.—**The Heart.**—Palpation confirms inspection as to the shape of the precordia, the position and the extent of the impulse, and the condition of the intercostal spaces. In addition, we determine by palpation the regularity, character, and strength of the *impulse* and the presence or absence of *valve shock*, *thrills*, or *friction*. Palpation also reveals edema of the surface and fluctuation.

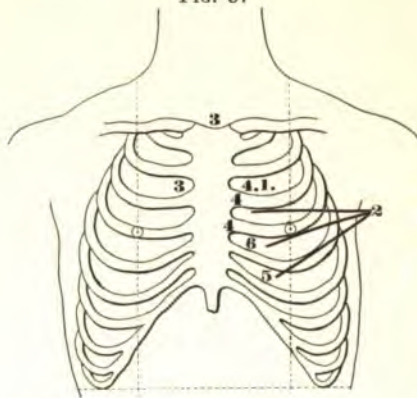
THE CARDIAC IMPULSE.—In a normal chest with moderately thick walls, a slightly prolonged, moderately strong shock is transmitted to the hand when placed lightly over the precordia. It is synchronous with the cardiac systole and precedes the radial pulse. It is therefore systolic in time. It is stronger when the patient leans forward and exhales freely, removing the lung from the surface, and when the chest walls are thin; it is weaker in opposite conditions.

Character and Strength of the Cardiac Impulse.—The force of the beat varies much in health. It seems increased in force in physical disturbances, bodily exertion, so-called cardiac neuroses, in some conditions that increase the rapidity of the heart's action, such as fevers,

and in the conditions that have already been detailed as increasing the extent of the impulse.

True increase in force of the impulse is seen in disease of the heart. When the organ is hypertrophied or the seat of dilated hypertrophy, the force of the impulse is increased, sometimes to an almost unbearable degree. Uplifting of the precordial area or even of the lower half the anterior part of the chest is seen. The hand or the head laid over the heart is forcibly lifted with each systolic contraction. This great force is most pronounced in the enormous hypertrophy that occurs in cases of aortic regurgitation or in endarteritis. It is the impulse and force of the so-called *cor bovinum*. In dilatation the impulse is diffused and wavy. The impulse may be diminished in strength or entirely absent: (a) in conditions apart from the heart on

FIG. 57



Abnormal palpable impulse and thrills. 1, diastolic impulse palpable from closure of pulmonic valve; 2, presystolic impulse often seen in third, fourth, and fifth interspaces in mitral obstruction; 3, thrill at aortic orifice: systolic, obstruction; diastolic, regurgitation; 4, thrill at pulmonary orifice: systolic, obstruction; diastolic regurgitation; 5, thrill at mitral orifice: systolic, regurgitation; diastolic, obstruction presystolic, obstruction; 6, thrill at tricuspid orifice.

account of which something intervenes between the heart and the chest wall; hence in general emphysema of the lungs and in compensatory emphysema of the left lung the impulse is entirely effaced. General debility, fainting and collapse are conditions when the impulse may not be felt on account of the lowness of the blood-pressure. (b) In *disease of the pericardium* the impulse is absent when there is large effusion; the absence here succeeds the dislocation to the left, and with its effacement the impulse in the second and third interspaces disappears. (c) In *disease of the heart* the impulse is absent when the heart is diminished in size, as in atrophy, or when weakened by any cause.

We must bear in mind that the cardiac impulse may be entirely absent in health, and too much importance should therefore not be attached to its absence.

THRILLS.—A thrill is produced when the blood is thrown into vibration by passing over a rough surface. It can be created only at the time blood is passing through the orifices, and increases as the force of the stream increases. (1) The most common seat of the thrill is the apex. If the hand is placed in close proximity to the surface of the chest at this point, a purring vibration or tremor is transmitted to it in most cases of *mitral obstruction*. The blood is passing from the auricle to the ventricle; as this takes place before the systole, the thrill is felt before the impulse or carotid pulse and is *presystolic* in time. It is sometimes difficult, however, to distinguish it from the impulse. The vibrations or the thrill is clearly transmitted to the hand. Sometimes the thrill continues throughout the entire diastole. (2) The next most frequent seat of thrill is over the aortic area and is caused by obstruction at the aortic orifice. It may be felt away from the heart, in the aorta, or in the carotids. This systolic thrill must not be confounded with the thrill elicited over the aorta or at the aortic cartilage in aneurism. (3) Sometimes a thrill is felt at the apex with the systole—*first sound*. This must not be confounded with the before-first-sound thrill. It is never so distinct, and is not made up of a series of vibrations. It is due to regurgitation at the mitral orifice. (4) Rarely a thrill is felt at the second cartilage on the right, with the second sound. It may be felt along the course of the sternum also, and is due to regurgitation through the aortic orifice. (5) At the second costal cartilage on the left a thrill is sometimes felt. It is systolic in time and is not transmitted. It is due to obstruction at the pulmonary orifice. (6) At the lower portion of the sternum a thrill, systolic in time, may also be felt, due to tricuspid regurgitation. Care must be taken not to confound the above-mentioned thrills with those due to aneurism. (See Aneurism.)

PERICARDIAL FRICTION.—A friction or to-and-fro rubbing is transmitted to the hand in cases of pericarditis in the fibrinous stage. The friction may be felt all over the heart region, but is pronounced in the third or fourth interspace. It may be detected on slight pressure or only when the tips of the fingers are pressed firmly against the interspaces.

It is important to remember that the position of the patient weakens or modifies the thrill or friction. When the patient is lying down it may not be felt. The upright posture or leaning forward makes it evident, and hence the patient should be instructed to assume this position in the examination.

The Arteries.—The results of inspection are confirmed. In addition the artery is examined to determine its tension, the condition of the coats and the presence of thrills.

In examining the arteries it is important, as will be detailed in the section devoted to the pulse, to compare the arteries of the two sides. Often the pulses are found to be unequal in force, in volume, and in time. This is almost always due to obstruction to the passage of the blood. When not due to *endarteritis* or aneurism it is due to the

pressure of a tumor on the vessel somewhere in its course, or to a thrombus or embolus in the artery. A difference between the radial and the femoral pulse points to obstruction in the thoracic or abdominal aorta. Anatomical variations must be remembered.

The Pulse.—The pulse is an index of the force, frequency, and rhythm of the heart's action and of the pressure or tension which is maintained in the arteries.

GENERAL OBSERVATIONS.—The frequency of the pulse before birth is from 120 to 140 beats per minute. From this time it is diminished in frequency up to adult life, 72 being then accepted as an average; the number of beats, however, is often more and sometimes less than 72. In old age the pulse-rate is again increased. Sex has some influence. The rate is slightly higher in females than in males of the same age.

The frequency of the pulse is subject to diurnal variations, at times corresponding with the diurnal rise and fall of temperatures. The rate will, therefore, be highest in the afternoon and evening and lowest in the early morning hours.

The position of the body also has a modifying influence. The pulse is more frequent when a person is standing than when he is sitting, and more frequent when he is sitting than when he is lying down. Walking, running, bodily and mental exertion, fear, and excitement all tend to accelerate the pulse.

During and for one or two hours after a meal the pulse-rate is higher, especially if an alcoholic or other stimulant, such as coffee, has been taken.

HOW TO TAKE THE PULSE.—To make a correct count of the frequency of the pulse, the conditions just mentioned as normally modifying its rate should be borne in mind, and every effort made to avoid extraneous exciting influences. In hospital practice, or when a nurse is constantly in attendance, the pulse and respiration should be taken at the same time as the temperature, and when the patient is undisturbed and quiet.

The wrist is the place usually selected for feeling the pulse. At this point the radial artery passes over the radius, and can readily be compressed and its character made out. In particular cases it may be advisable to count the pulse at the temporal or carotid artery. Three fingers should be applied lightly so that the beats can be most distinctly felt. The beats are counted for fifteen seconds by the second hand of a watch when only an approximate count is desired or when time is a factor, and then multiplied by four. It is better, however, to count the pulse for half a minute, and still better for a full minute.

The arteries of the two sides must be compared. Differences in force, volume, and time may be due to anomalous distribution of arteries. In disease such differences may occur in aneurism and atheroma, in pressure on the trunk from external disease, and in embolism and thrombosis.

CONDITION OF THE WALLS OF THE ARTERY.—The condition of the artery is often of more importance than the pulse-rate. A healthy

radial artery in a person not advanced in years can be compressed easily against the radius without the finger being able to differentiate the artery from the other tissues. In the aged, or in arteriosclerotic individuals from whatever cause, the artery is thickened and in pronounced cases unobliterable, rolling like a pipe-stem under the palpating fingers. In advanced cases of arteriosclerosis irregular calcareous deposits are palpable in the arterial wall.

VOLUME.—The volume of the pulse refers to the expansion of the artery during each beat. It is usually large in conditions of pyrexia and when the tension is low. A small pulse is met with in many conditions other than weakness of the heart muscle. In aortic stenosis the pulse is of small volume and in mitral stenosis it is small, of high tension, and frequently irregular. In Bright's disease it is sometimes very small, tardy, and hard. Some care will be required to differentiate such a pulse from a weak pulse. In acute peritonitis the pulse is apt to be of small volume and low tension.

CELERITY.—A pulse is said to be "quick" when it rises rapidly, the wave lightly hitting the palpating finger and immediately disappearing. A pulse of this character is characteristic of conditions of low blood-pressure, but is typically exemplified by the water-hammer or Corrigan pulse of aortic insufficiency. It is usually a pulse of large volume. The tardy pulse is one that seems to slowly strike the finger and as gradually disappear. It is common in all conditions of high pressure and is generally of small volume.

A *dicrotic pulse*, recognized by the occurrence of a small secondary pulse wave after the primary wave, is common in conditions in which the pulse is quick and not of good volume, that is when there is a general, relaxation of the peripheral vessels. It can frequently be felt in normal individuals of low blood pressure or in prolonged febrile conditions, notably typhoid fever in the second and third week.

RHYTHM.—The rhythm of the pulse is of diagnostic importance. In health one beat succeeds another at equal intervals of time, and the successive beats are of equal force and like quality. When there is irregularity there may be alternation in the rhythm of the pulse or in rhythm and in the force. (See Irregularities.)

FREQUENCY.—The frequency of the pulse is of aid in diagnosis.

Increased Frequency.—The pulse is increased in frequency in practically all febrile diseases, and generally in the proportion of 8 to 10 beats for each degree of rise in temperature above 98.6°. But there are important exceptions. In *typhoid fever* the pulse is slower in proportion to the temperature and the gravity of the disease than in most of the other acute febrile diseases. It may not beat above 85 in mild cases, and in severe cases frequently does not rise above 100. It may be more frequent during convalescence than during the febrile stage. The pulse of *scarlet fever* often aids materially in diagnosis. A pulse of 120 to 160 is the rule from the time of development of the sore throat to the completion of the eruption. In incipient tuberculosis

the increased pulse-rate noted is a valuable confirmatory finding. In the puerperium increased frequency of the pulse is a surer indication of intra-uterine mischief than is the temperature. So, too, in all cases of inflammation so situated that the products are absorbed into the circulation and not discharged externally, the pulse shows by its increased frequency that a septic process is going on.

In Graves' disease great frequency of the pulse is the essential and most constant symptom. The pulse may be constantly much more frequent than 100, and in attacks of palpitation the number of beats may reach 200 or more. *Paroxysmal tachycardia* is characterized by the sudden onset of a marked increase in pulse-rate persisting for an indefinite time and disappearing almost as suddenly as it came. In shock or collapse, in central or peripheral vagus disease, in many nervous diseases, *e. g.*, locomotor ataxia, in anemic conditions, Addison's disease and in arthritis deformans, the pulse is increased. Malingering may be differentiated from conditions in which there is real pain by the occurrence of a rapid pulse in the latter case.

Overindulgence in alcohol, tobacco, tea or coffee, overwork, exhaustion, insomnia, and mental strain may all cause increase in the heart-rate. Increase of pulse-rate is common in all acute cardiac conditions and in chronic cardiac conditions from whatever cause when there is myocardial insufficiency.

Increase of pulse-rate upon slight exertion or excitement develops as a result of pressure on the heart by pleural effusions, ascites, tympanitis, etc., and when there is impairment of functional power of the heart from overstrain, myocardial disease, during convalescence from fevers, in anemic, asthenic, or cachectic conditions, etc.

Diminished Frequency.—A slow pulse (bradycardia), under 60, like a frequent pulse, is sometimes habitual and sometimes a family characteristic. A slow pulse is met with in certain forms of heart disease, as aortic stenosis, but it is not constant in any of them except in heart-block. It occurs in fatty degeneration, especially when due to obstruction, by atheroma or otherwise, of the coronary arteries. When retardation appears in the late stages of cardiac affections or specific diseases with cerebral symptoms, it is usually a sign of danger. Bradycardia is constantly seen in articular rheumatism, usually always in jaundice and frequently in lead poisoning. It is most common in convalescence from acute diseases, particularly pneumonia, typhoid fever, erysipelas and rheumatic fever, and in cachectic, asthenic, and exhaustive conditions. It is also frequently encountered in diseases of the digestive organs and of the urinary organs, particularly acute nephritis. Moreover, the pulse is generally slow in myxedema, and in epilepsy. Not uncommonly it is slow also in melancholia, during the early stages of cerebral meningitis, especially when tuberculous, and in cerebral tumors and hemorrhage.

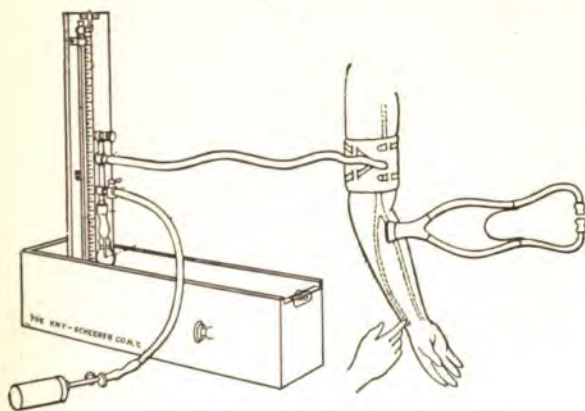
The Blood-pressure.—The tension or pressure of the blood within the arteries is known as the blood-pressure. The exact estimation of

the blood-pressure is of the greatest value, not only in diagnosis, but also in prognosis and treatment and can only be properly performed with an instrument of precision—the *sphygmomanometer*. The ease of use of this instrument and the vast amount of information derived from its use make it one of the most valuable instruments that a physician can employ.

Apparatus.—Numerous sphygmomanometers have been devised, but for accuracy and convenience combined the mercurial instruments have been found to far surpass the other varieties. The spring instruments are inaccurate, inasmuch as the elasticity of the spring varies considerably and sooner or later wears out entirely. Instruments using water columns to register the blood-pressure are too inconvenient for ordinary use, owing to the length of the manometer tube. The instruments using the mercurial column have been so perfected that they are extremely accurate and at the same time are convenient and portable. Many types of this sphygmomanometer are on the market, but the “Nicholson” amply fulfils all requirements.

The older model is extremely satisfactory but Nicholson has recently brought out a newer model which is smaller and consequently more portable but as accurate and convenient as the older model.

FIG. 58



Auscultatory method of determining blood-pressure.

METHOD OF DETERMINING BLOOD-PRESSURE.—The apparatus is made ready for use and the cuff fitted to the bare upper arm, which should be at the level of the heart. In children the thigh may be employed with the patient in the prone position. The cuff is inflated until the radial artery is obliterated at the wrist. The bell of a biurnal stethoscope is then placed lightly over the brachial artery in the bend of the elbow. It is well to first mark the skin overlying the artery with a dot of ink so that the stethoscope will be placed directly over the

artery, which cannot be palpated, as it has been obliterated by the pressure of the cuff. The air is now permitted to slowly escape from the cuff. As it escapes the column of mercury gradually falls and a loud clear thump can be heard by the listener when the first complete cardiac beat passes the constricting cuff. When this thump is heard the height of the mercury column is read off and these figures represent in millimeters of mercury the *systolic pressure* or the greatest pressure exerted by the heart during the systole. The bell of the stethoscope is kept over the artery, and as the air escapes from the cuff the loud clear sound (the first phase) is followed by a murmur (the second phase) which in turn is replaced by a clear loud ringing thump (the third phase) which gradually becomes duller (the fourth phase) to disappear entirely (the fifth phase). The *diastolic pressure*, or lowest pressure during the cardiac cycle, occurs at the end of diastole, and is estimated by reading the height of the mercurial column when the second loud thump becomes dulled, *i. e.*, the beginning of the fourth phase. The meaning of the various phases has not as yet been interpreted, though the intensity of the third phase is supposed to be indicative of the cardiac strength. The *pulse-pressure*, the difference between systolic and diastolic pressure, is found by subtracting the diastolic pressure from the systolic pressure. This method of estimating the blood-pressure, the auscultatory method of Korotkow, is so accurate, in fact is the only accurate method of estimating the diastolic pressure, that it should always be employed and the older palpation method should be discarded.

PHYSIOLOGY OF BLOOD-PRESSURE.—Normal pressure depends upon four factors: (1) the contraction of the left ventricle; (2) the resistance in the peripheral vessels; (3) the elasticity of the arterial walls; (4) the volume and fluidity of the blood.

During ventricular systole the blood driven out by the left ventricle reaches its greatest pressure in the aorta and soon afterward the aortic valves close. Pressure is then sustained until the next systole by the elasticity of the vessel walls, but it gradually falls, as the blood enters the capillaries, to reach its lowest point at the end of diastole. The pressure is thus seen to depend largely upon the strength of the cardiac beat and the peripheral resistance which in turn depends upon the contractility, distensibility, and caliber of the vessel walls. It follows then that the systole, or maximum pressure, shows the strength of the cardiac beat, while the diastolic, or minimum pressure, is indicative of the peripheral resistance and the pulse-pressure of the head of pressure, driving the blood from the heart through the large vessels to the arterioles. The practical applicability of these facts may be gathered from the following: A weak heart is unable to raise the blood-pressure, so the systolic pressure is low. If the diastolic pressure is also low, *i. e.*, a high pulse-pressure, presumably there is an associated dilatation of the peripheral vessels. This may be so great, as in the condition of shock in which the heart may be strong, yet from the lack of peripheral resistance

from vasomotor paralysis it is as if the heart were pumping against nothing, and the systolic pressure cannot be raised by the ventricular stroke. Conversely a low systolic pressure with a low or slight pulse-pressure surely indicates a weak heart, as the peripheral resistance is normal, though the vessels may possibly be slightly dilated. High systolic and diastolic pressure indicates strength of the heart-beat with corresponding increase in the peripheral resistance. A high systolic pressure with a low diastolic pressure, *i. e.*, a large pulse-pressure, shows a powerful heart (hypertrophied) pumping against relaxed vessels. Furthermore, an increase of pulse-pressure generally indicates increased velocity of blood flow (pulse-pressure shows the head of flow) if the pulse is constant, and the converse also holds true as a rule.

NORMAL PRESSURE.—The normal systolic pressure in adults before midlife varies between 110 mm. and 130 mm. Deviations of 10 mm. of mercury either above or below these figures can hardly be called pathological, but are certainly suspicious. Variations above 140 mm. or below 100 mm. are distinctly pathological. The readings in those past midlife are higher than in earlier adult life, so that a systolic pressure up to 150 mm. can hardly be called abnormal or pathological. In women the pressure usually averages about 10 mm. lower than in males, while children show even lower pressures. The diastolic pressure is from 30 to 45 mm. Hg. (Hirschfelder) lower than the systolic pressure, that is, the average pulse-pressure is between 30 and 45 mm. As a rule the diastolic pressure does not increase, *pari passu*, with the systolic pressure, so that usually the pulse-pressure is increased when there is high systolic pressure.

PHYSIOLOGICAL CONDITIONS AFFECTING THE BLOOD-PRESSURE.—*Sleep* lowers the systolic pressure from 10 to 20 mm. and the diastolic pressure to an even greater extent. *Eating* causes a moderate rise in systolic and a smaller rise in diastolic pressure. *Deep breathing* raises blood-pressure during expiration, lowers it during inspiration. *Moderate exercise* has the same effect as eating, only more marked. *Excessive exercise*, to the point of fatigue, lowers the systolic pressure. *High attitudes* cause a rise in pressure. *Pain, anger, excitement, emotion, mental strain, and worry* all cause a rise in systolic and a still greater proportional rise in diastolic pressure. *Intense pain* produces shock with an associated fall in pressure. The position of the patient causes changes in the blood pressure, thus while reclining the pressure is 8 to 10 mm. higher than while sitting. Continued constriction of the brachial artery by the cuff causes a rise of pressure up to 20 mm. Hg.

PATHOLOGICAL CONDITIONS AFFECTING THE BLOOD-PRESSURE.—**HYPERTENSION.**—It is of primary importance to remember that persistence of high pressure is maintained only with cardiac hypertrophy and that persistent high pressure causes arteriosclerotic changes. It is also well to remember that a heart which is apparently *grossly* weak may give a relatively high systolic pressure; such pressure may be low and insufficient for that individual whose organism requires and has

been accustomed to high pressure from some cause. Thus cases of advanced cardio-renal disease with relatively high pressure (in whom the pressure had previously been continuously much higher for a long time) are frequently seen, but such pressure, though high, is insufficient. Furthermore, it is frequently observed that in decompensated cardiac cases the pressure is higher than when compensation is restored. Such heightened pressure in myocardial insufficiency is explained by slowing of the circulation with consequent asphyxia of the medullary centres, causing a marked vasoconstriction which requires increased effort on the part of the heart to overcome with consequent increase in the systolic pressure.

The following conditions are associated with high systolic pressure:

1. *Nephritis*.—In the various types of this disorder, differences in the pressure are marked. Hypertension in *chronic interstitial nephritis* is always present unless there is pronounced myocardial weakness, the figures for the systolic pressure usually being over 200 mm. with a relatively low diastolic pressure. High pressure is an extremely valuable diagnostic sign of this condition which may be diagnosticated on this finding alone, in the absence of any other demonstrable cause for the high pressure. *Chronic parenchymatous nephritis* usually also causes a high pressure but not as regularly as the chronic interstitial form, as frequently chronic cases of the parenchymatous type of the disease are seen with low pressure. In *acute nephritis* the pressure is usually low. In the acute form occurring in scarlatina (glomerulonephritis) the onset of clinical symptoms is usually preceded by a sharp rise in systolic pressure of 30 to 50 mm., a valuable diagnostic and prognostic sign. In *uremia* the blood-pressure is high, the rise of pressure running parallel with the severity of the attack, and falling with the disappearance of symptoms or with failure of the heart.

2. *Increased Intracranial Tension*.—The highest pressures known occur in conditions which give rise to increased pressure within the cranial vault and are indicative of cerebral anemia, with consequent need of the brain for blood. The conditions causing this extremely high pressure are apoplexy, thrombosis, intracranial hemorrhage, fracture of the skull, meningitis, cortical epilepsy, and rapidly growing cerebral neoplasms.

3. *Heart Conditions*. With hypertrophy of the left ventricle there is always an increase of systolic pressure (140 to 170 mm. systolic, 90 to 110 diastolic) if not influenced by other factors. The great hypertrophy of the left ventricle together with the usually associated arteriosclerosis account for the high pressure (170 to 200 mm. systolic, 90 to 120 mm. diastolic) commonly seen in aortic insufficiency, a condition always associated with a high pulse-pressure and in about 80 per cent. of cases with an absence of the fifth auscultatory phase. Other valvular lesions are usually associated with normal or slightly increased pressure if there is marked left ventricular hypertrophy and good compensation. Cardiac arrhythmias are usually associated with

normal pressure, and an associated high pressure is usually of grave prognostic significance. As said before, decompensated cardiac conditions usually show a high systolic pressure.

4. *Disease of the Arteries.*—The close association of arteriosclerosis with cardiac hypertrophy and chronic nephritis is sufficient in most cases to account for the high pressure seen in sclerotic persons. In arteriosclerotic individuals without complicating nephritis the systolic pressure will usually average between 150 to 170 mm., the diastolic between 90 to 100 mm. However many cases of primary arteriosclerosis are without high pressure which seems to depend entirely upon sclerosis of the splanchnic vessels.

5. *Angina Pectoris.*—Attacks of angina are often associated with high pressure, but on the contrary in many cases the pressure is normal or lowered during the attack.

6. *Miscellaneous Conditions.*—Lead-poisoning (acute or chronic) and gout are usually accompanied by hypertension. Chronic polycythemia and leukemia, if there is no anemia, on account of the increased viscosity of the blood with consequent increased cardiac effort, are associated with heightened blood-pressure. Exophthalmic goitre is also associated with high pressure if there is hypertrophy of the heart.

HYPOTENSION.—1. *Chronic Pulmonary Tuberculosis.*—This disease in uncomplicated cases is probably always associated with low systolic pressure, *i. e.*, at least under 110 mm. Hg. and in the majority of cases between 90 and 100 mm. The diagnostic importance of these findings in suspected incipient cases cannot be too strongly emphasized when extraneous causes of low pressure can be ruled out. Furthermore, in cases of known tuberculosis a falling pressure is indicative of continued progress of the disease; a rising pressure shows that the patient is improving.

2. *Acute Cardiac Conditions.*—The systolic pressure in acute cardiac conditions not preceded by chronic cardiac disease or accompanied by marked cyanosis, is usually markedly lowered. This applies to acute endocarditis, myocarditis, and more particularly acute pericarditis or pericarditis with effusion from whatever cause.

3. *Cachectic Conditions.*—Cancer, severe anemias, tuberculosis of any organ or part, chronic malaria, etc., are all associated with a systolic pressure of from 5 to 20 mm. lower than the normal.

4. *Acute Infectious Diseases.*—In practically all the acute infectious diseases, excepting meningitis, the systolic pressure is usually below 100 mm. Hg. when the fever is at its height. In *typhoid fever* the fall is gradual, reaching its lowest point during the last two weeks of the disease and frequently not exceeding a maximal pressure of 80 mm., or even lower. The blood-pressure estimations are frequently of value in diagnosing the complications of this disease. With hemorrhage there is often a brisk sudden fall; with perforation, on the contrary, the systolic pressure in a certain number of cases is slightly raised. *Pneumonia* is associated usually with a pressure slightly subnormal.

Gibson has called attention to a valuable prognostic sign: a systolic pressure higher than the pulse-rate is of good omen, while, on the contrary, a systolic pressure below the pulse-rate is indicative of a serious condition of the patient. Furthermore, the observation of the pulse-pressure is of importance in treatment, a high systolic pressure with low diastolic pressure is indicative of vasomotor weakness, while the opposite findings show that heart failure is imminent. In scarlet fever, as said before, a sharp increase of systolic pressure may be the first indication of a complicating nephritis.

5. *Severe Hemorrhage and Diseases Associated with Loss of Body Fluids.*—In these conditions the lowered systolic pressure is the direct mechanical result of lack of blood necessary to fill the arteries. The pressure in the first condition usually rises after controlling the bleeding. In the second condition, which include dysentery, cholera, and severe vomiting, as in cancer of the stomach, peritonitis and intestinal obstruction, the lower the pressure falls the greater the danger of collapse.

6. *Shock (Collapse).*—In this condition an extremely marked fall of pressure takes place as a result of vasomotor paralysis. Maximal pressures as low as 50 and even 40 mm. have been recorded.

7. *Splanchnoptosis.*—In individuals with general ptosis of the abdominal viscera the maximal pressure is usually markedly lowered, probably the direct result of relaxation and dilatation of the abdominal vessels. In a certain percentage of patients who are apparently the victims of neurasthenia, nothing abnormal can be found except a low systolic blood pressure and not even ptosis of the abdominal organs can be demonstrated. The symptoms in many such cases are unquestionably due to the lowering of the blood pressure as a result possibly of dilatation of the splanchnics from the general lack of tone or as a result possibly of congenitally overlarge abdominal vessels with consequent insufficiency of the peripheral circulation. It is astonishing to find how many so-called neurasthenics there are with lowered blood pressure.

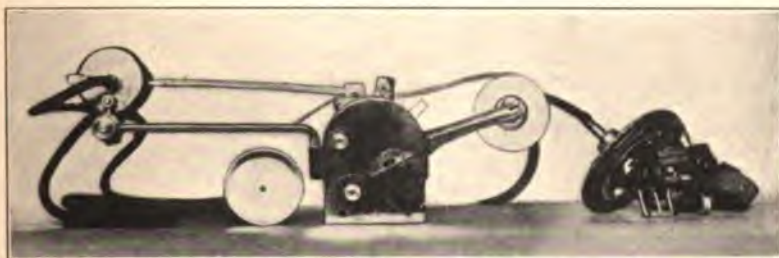
8. *Miscellaneous Conditions.*—Serofibrinous pleurisy is usually associated with low blood pressure. In delirium tremens some cases of insomnia, acute mania, and hysteria the pressure is uniformly lowered. In the acute stages of syphilis the pressure is lowered. In parasymphilitic affections the pressure varies; thus in the late stages of general paresis there is hypotension present; in tabes, during the lightning pains, hypotension is also noted while in gastric crises the pressure is markedly raised, a point of value in differential diagnosis, as the colic of plumbi m and abdominal angina are the only other two abdominal disorders associated with high pressure. Acute inflammatory intra-abdominal conditions are usually associated with low pressure as a result of the dilatation of the abdominal vessels from the resulting local inflammation.

GRAPHIC RECORDS OF THE PULSE.—The study of the arterial pulse as outlined above yields information which only informs us what the

left ventricle is doing. To accurately study what the other cardiac chambers are doing it is necessary to make simultaneous tracings of the arterial and venous pulsations. By comparing the graphic records thus produced, knowledge is obtained concerning the efficacy of the inherent properties of the heart muscle (*q. v.*), and abnormalities in these properties can be appreciated.

The Polygraph.—Sphygmographic tracings of the arterial pulse have been employed for a long while to illustrate various forms of pulse waves, and diagnostic information as to volume, force, and tension of the pulse is supposed to be obtainable from these tracings; but, as a matter of fact, no real knowledge can be gotten from such tracings, except in regard to irregularities, which, however, are only accurately studied by the polygraph, as the character of a pulse-wave can be very materially altered and various forms and modifications of pulse-waves can be easily made upon the same person by moving the instrument, changing the pressure, and other manipulations. The polygraph is

FIG. 59



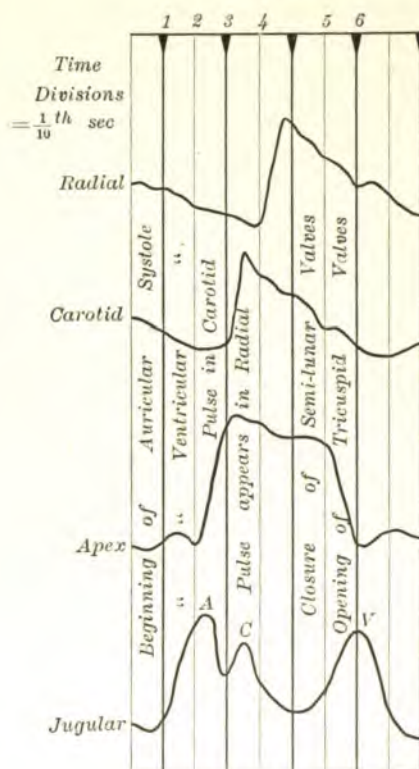
Mackenzie's ink polygraph.

an instrument devised for the purpose of making simultaneous venous, carotid, apical, or radial registrations. Various types of polygraphs have been devised, but for convenience, ease of application, portability, and because prolonged tracings can be made, the Mackenzie ink polygraph probably surpasses all others for ordinary clinical work. This instrument consists of a clock which moves an encased roller at controllable speeds and a time marker which records 0.2 second by means of a pen and marker. Behind the instrument is an arm supporting a roll of paper the end of which is carried to the writing platform and moved forward by the roller beneath. In front there is a socket to attach a lever to which in turn are attached two tambours. To these tambours two long aluminum pens, with receptacles for the ink, are inserted, and when ready for the tracing these are adjusted lightly on the paper. Rubber tubes connect the tambours with two metal cups, in order to take carotid or apical tracings at the same time as the jugular, or with one cup for the jugular and with the radial receiver, which is strapped on

the wrist and which can be accurately adjusted upon the artery. Red ink is placed on the pens, as it is unlikely to clot.

When a tracing is to be made the clock is wound up, the radial receiver adjusted so that a maximal amount of movement is obtained by the connected lever, and the jugular cup placed lightly, and with only sufficient pressure to exclude the air, upon the jugular vein as near the jugular bulb as possible. The long levers are lightly adjusted to the paper and the clockwork started and allowed to run as long as desired.

FIG. 60

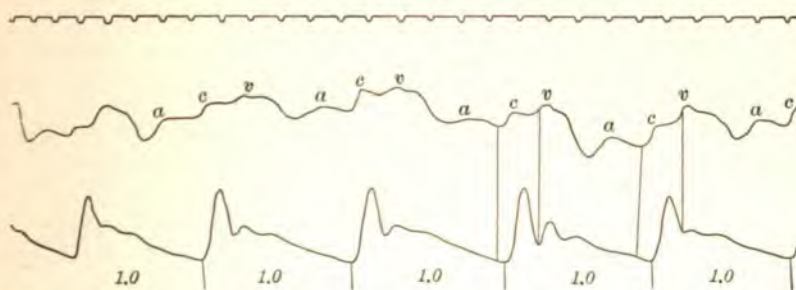


Composite drawings of curves of apex-beat and radial, carotid, and jugular pulses.
(Whiting.)

Interpretation of the Tracing.—By referring to Fig. 60 it can be seen that the beginning of ventricular systole in the apical tracing (the graphic representation of the movements of the chest wall) is shown by the beginning of the up-stroke. One-tenth of a second later than this the up-stroke in the carotid commences and one-tenth of a second later than this the radial up-stroke. The one-tenth second before the carotid up-stroke is known as the pre-sphygmic period, *i. e.*,

the period in which the ventricle contracts without impression on the arterial pulse. Just preceding the ventricular up-stroke is a small wave, produced by the distention of the ventricle by the blood pumped in during auricular systole. Upon the down-stroke of the carotid and radial tracings there can be seen a depression, the dicrotic notch, marking the end of ventricular systole. In the venous tracing three main waves are seen, the *a*, *c*, and *v* waves, respectively representing the wave (*a*) due to auricular contraction, the wave (*c*) occurring at the onset of ventricular contraction either caused in part by the pushing up of the tricuspid valve when the intraventricular pressure rises and in part by the flow of blood from the coronary veins (Hirschfelder) or else due to the impact of the carotid expansion on the near-by jugular, and the wave (*v*) due to the stasis of blood during diastole in the auricle and jugular bulb. At the end of ventricular systole the auriculoventricular valves open, the blood rushes into the ventricles, and the wave is followed by a fall as the blood empties from the veins.

FIG. 61



Normal jugular and radial pulse, from a case of aortic insufficiency. *a*, wave due to auricular systole; *c*, wave due to impulse of carotid; *v*, stasis wave.

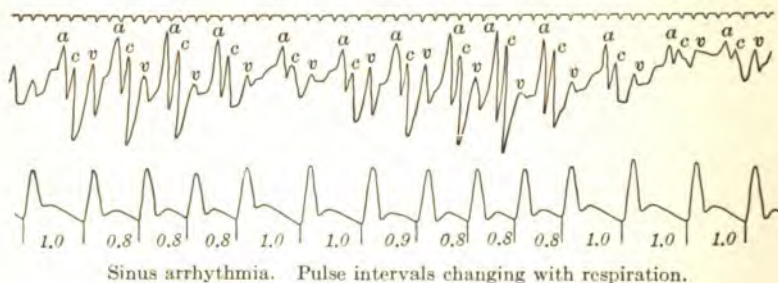
Analysis of the Tracings.—When a tracing has been made it is necessary to identify the various jugular waves that have been made. This is done by comparing them with the radial tracing, and it is performed as follows. The *c* wave occurs synchronously with the pulsation of the carotid or $\frac{1}{10}$ second before the radial. Therefore $\frac{1}{10}$ of a second or one-half of the division of the time record is measured off with a pair of calipers. This distance is then transferred to the radial tracing so as to immediately precede the beginning of the up-stroke. From this point measurement is made to the line made at the beginning or end of the tracing by the movements of the lever before starting or after stopping the clockwork—the ordinate. This distance is then transferred to the jugular tracing and will mark the beginning of the *c* wave. Better still is a simultaneous tracing of the carotid and jugular, at the same level in the neck, as the *c* wave can then be determined absolutely without the necessity of such exact and fine measuring. The *a* wave is obtained by measuring a distance of $\frac{1}{6}$ second (one

division of the marker) back from the *c* wave as the interval between the beginning of the auricular contraction and the carotid pulse is $\frac{1}{2}$ second. The *v* wave is obtained by measuring the distance from the ordinate to the dicrotic notch and transferring this measurement to the jugular tracing. A tracing of this form is the normal and is known as the negative or auricular form of venous pulse. When there is auricular fibrillation a form of venous pulse occurs which is known as the ventricular or positive venous pulse. In this form the auricle is fibrillating, so that no pulsation of this heart chamber takes place and consequently there is entire absence of an *a* wave. The *a* wave is also absent in tricuspid insufficiency as the right heart is over-distended, and in simultaneous contraction of the auricle and ventricle. In the latter cases the arterial pulse is regular, while in the former absolutely irregular.

VISIBLE VENOUS PULSATIONS.—Visually the *a* and *v* waves can be sometimes appreciated as two pulsations of the jugular between each carotid pulsation which is felt by the finger on the artery. In auricular fibrillation only one pulsation occurs between each carotid pulsation. The visible examination is satisfactory in only a few frank cases.

IRREGULARITIES OF THE PULSE.—The character of the irregularities of the heart can usually be determined by palpation of the pulse and by auscultation of the heart, but they are best studied by means of polygraph tracings of the pulse. For that reason they will be considered in this section. Various forms of irregularities have been described, but the following types include practically all the forms of irregularity of rhythm that are met with clinically. They include: (1) sinus arrhythmia; (2) extrasystoles; (3) alternating pulse; (4) absolutely irregular pulse; (5) heart-block.

FIG. 62



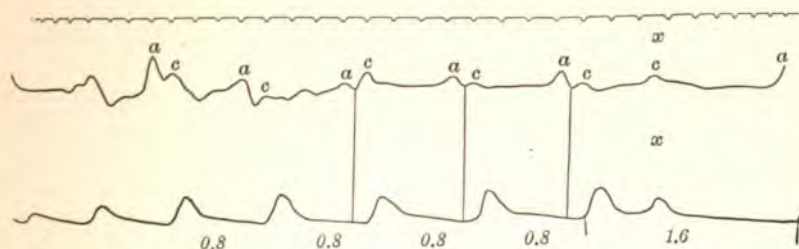
Sinus arrhythmia. Pulse intervals changing with respiration.

1. *Sinus Arrhythmia (Juvenile Respiratory Arrhythmia).*—Arrhythmia of this type is characterized by rhythmical slowing of the diastole during the act of respiration. Such slowing may occur during either inspiration or expiration, and at times during swallowing. A pulse of this type is the result of reflex vagal stimulation, which slows the heart. It is usually readily recognized clinically, as it is exaggerated by deep

breathing. Sinus arrhythmia occurs in children, neurotic and nervous individuals from any cause, persons suffering from fear or anxiety, and after acute infections. This form of irregularity is without clinical importance, and certainly does not signify pathological changes in the heart, but signifies rather the contrary, as it shows the heart's contractility is unimpaired.

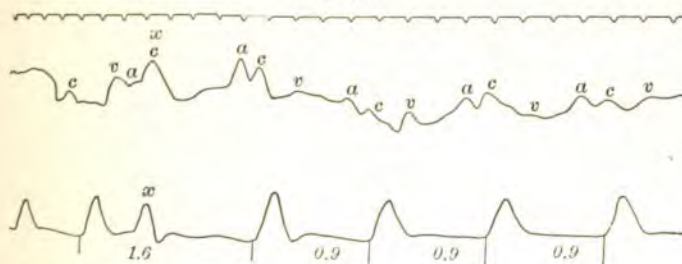
2. *Extrasystoles*.—These may arise as a premature contraction of the auricle or the ventricle. They are characterized by the interpolation of an extra heart-beat immediately following a regular beat. This accessory contraction causes a small pulse-wave immediately after the normal wave and is followed by a long diastole or compensatory

FIG. 63



Extrasystole marked by *x*, showing complete compensatory pause, i. e., the extrasystole and the previous beat are together equivalent in time to two normal beats.

FIG. 64



Extrasystole marked by *x*, showing incomplete compensatory pause, i. e., the extrasystole and preceding beat together are unequal to the sum of two normal beats. Also showing increased amplitude of pulse following extrasystole.

pulse and a more powerful systole than before. The extrasystoles may occur only at long intervals or they may occur more frequently, as after every third or every second beat (pulsus trigeminus or bigeminus). In some cases the extrasystole when occurring in early diastole is so weak as to fail to open the aortic valves, and no pulse is felt for a long period, and the heart appears to have dropped a beat, though it is merely a prolonged compensatory pulse. The extrasystoles may arise in the auricle, the auriculoventricular node or in the ventricles.

They are supposed to be due to increased excitability of the primitive cardiac tube (Mackenzie). The ventricular extrasystoles are characterized by the fact that in the arteriogram the period in which the extrasystole occurs plus the previous beat is equivalent to the time it takes for two normal systoles to occur, so that there is no disturbance in the dominant cardiac rhythm. In the phlebogram there is an exaggerated peak if the extrasystole occurs at the time of the expected auricular contraction, otherwise there is the regular sequence of *a* waves plus an extra *c* wave. The premature auricular contractions may or may not show a compensatory pause, and "it appears certain that if in the case of any premature contraction the pause fails to be compensatory, then such a beat has arisen in the auricle." (Lewis.) The arteriogram is similar to that of premature ventricular contractions except for the absence of the compensatory pause in the majority of cases. When the auricular extrasystole is early, there is an absence of the preceding *v* wave and increase in the amplitude of the *a* wave in the phlebogram, the two waves coinciding, otherwise the phlebogram of the extrasystole, with the *a* wave, followed by the *c* and *v* waves, is as in the normal. The simultaneous premature contraction of both auricle and ventricle may be diagnosticated if the *a* and *c* waves occur together or very close to each other.

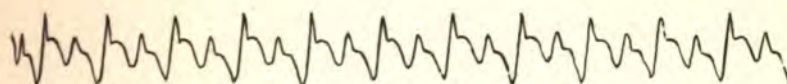
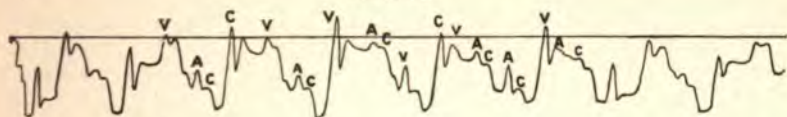
Clinically extrasystoles are usually readily appreciated without much difficulty. They occur notably in the aged, although they may be present in any acute or chronic cardiac condition, often in association with myocardial changes and hypertension. They occur also in neurotic individuals, in toxic conditions arising from the gastro-intestinal tract, from excessive coffee, tobacco, alcohol, etc., and in convalescence from acute affections, but are without much pathological significance. Subjectively patients sometimes feel as if the heart has stopped momentarily, or they may feel giddy for an instant.

3. *Alternating Pulse*.—The *pulsus alternans* is characterized by a regular succession of large and small pulse-beats. The genesis of this form of arrhythmia is unknown. It has been attributed to depression of contractility, to inaction of large portions of the ventricular musculature during alternate beats, and to heart-block in the ventricular wall. The condition is best recognized by the polygraph tracings. It occurs either in overtaxed hearts beating rapidly or in hearts in which there is pronounced changes in the musculature, as in advanced mitral stenosis, angina pectoris, fibroid heart, or after acute infectious diseases which have impaired the heart. From this it follows that it is of serious prognostic import.

4. *Absolutely Irregular Pulse (Perpetual Irregularity; Continuous Irregularity)*.—This type of pulse is characterized by irregularity in the force and equality of the heart-beats and in absence of any dominant rhythm. It occurs in the condition known as *auricular fibrillation*, in which the entire auricle is in continuous fibrillary contraction. As a result of this, multitudinous irregular stimuli originate in the auricle of

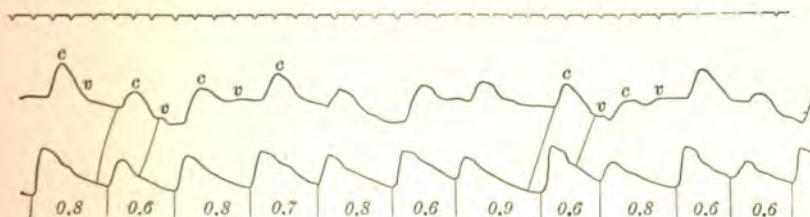
which only a few pass to the ventricle to cause its systole. The arterial pulse tracing shows an absolute irregularity of the pulse without any relationship to the length of the pause and the strength of the beat which follows. The phlebogram shows an absence of the *a* wave and is of the type of the positive venous pulse (*q. v.*).

FIG. 65



Pulsus alternans, from a case of cardiosclerosis suffering from angina pectoris and hypertension. This arrhythmia developed and was apparently induced by digitalis. It was most marked when the blood-pressure was high, as at the time the tracing was made. The patient died about three months later. (Norris.)

FIG. 66



Ventricular type of venous pulse, showing absence of a wave. Complete irregularity of radial arteriogram. From a case of auricular fibrillation with mitral stenosis.

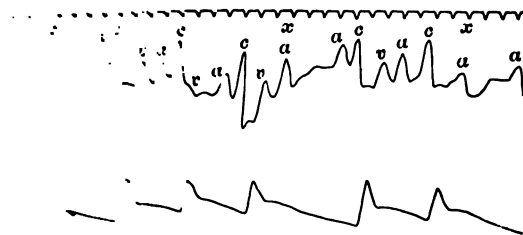
Auricular fibrillation is recognized clinically by the absolute irregularity of the pulse, by the presence in the neck of only one movement of the jugular between each carotid pulsation, at times by a humming sound heard on auscultation over the precordium, and by hearing systolic sounds the pulsations of some of which fail to reach the wrist. Lewis says that "in the vast majority of cases of complete irregularity of the heart auricular fibrillation is responsible for the disturbance of ventricular rhythm." It is responsible for at least 50 per cent. of all persistent irregularities of the heart. It occurs notably in mitral stenosis, but may occur in any cardiac condition

HEART-BLOCK

Heart-block is indicative of serious injury to the heart, and is the one heart condition upon which

the prognosis (in time) is characterized by a permanent or complete or complete. *Incomplete* heart-block is characterized by a prolongation of the interval between the onset of the auricular impulse; or (3) the failure of the auricular impulse (the so-called 3-to-1 rhythm). The characteristic tracings of the last two conditions in an electrocardiogram, because the auricular contraction is not followed by a ventricular contraction. *Complete* heart-block occurs, as the name implies, when the rhythm of the auricle and the rhythm of the ventricle are completely blocked and no

Fig. 67



tracing of a dropped beat in radial pulse with presence of a small r and r waves in jugular pulse. (Price.)

the condition of the auricle to the ventricle. The condition is often indicated by the polygraph tracings, but is also indicated by a slow pulse and, at times, by hearing at times a slow and dull distant sounds which are characteristic of the condition. Complete heart-block is frequently associated with Adams syndrome. The condition occurs in the bundle of His, as gummata, fibrosis, or other lesions involving all the myocardium, and is often seen to show a special predilection for the muscular tissues. Heart-block of a complete type is only in rheumatic cardiac affections and

The recent advances made in cardiac diagnosis have been rendered possible by the use of the electrocardiograph (the electrocardiograph). By the use of this instrument the preceding facts concerning arrhythmias have been absolutely demonstrated. It follows, there-

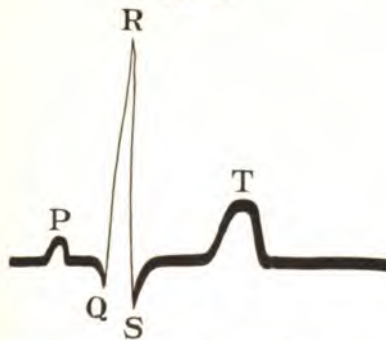
fore, that its employment is at times necessary to learn the true sequence of events in the cardiac cycle but on account of the extreme technicality of its management and the special skill required for the interpretation of the electrocardiographic curves, the use of this instrument should be left to the specialists in this branch of cardiology. The principle

FIG. 68



Leads attached to extremities. Impulses are carried from them, by the attached wires, to the instrument.

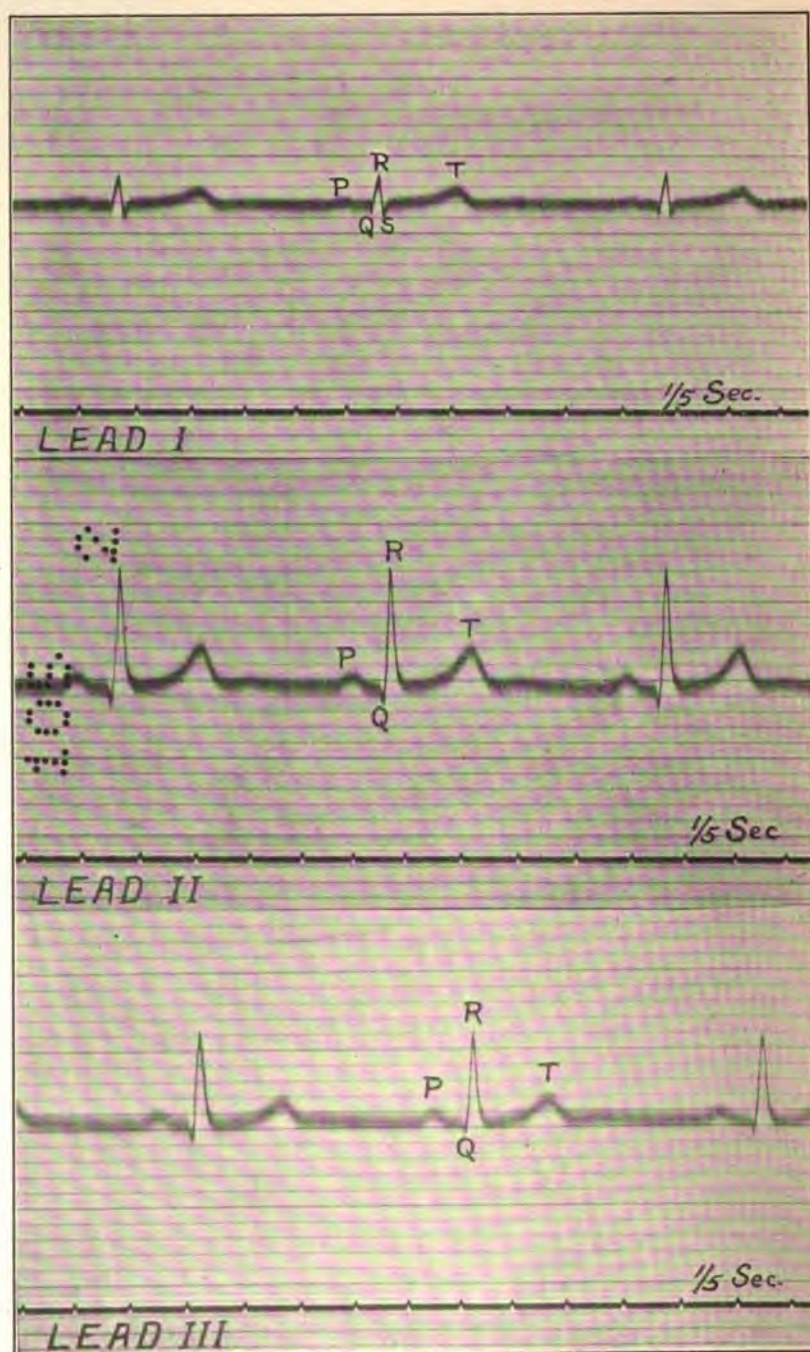
FIG. 69



Graphic representation of the electrocardiogram. (James.)

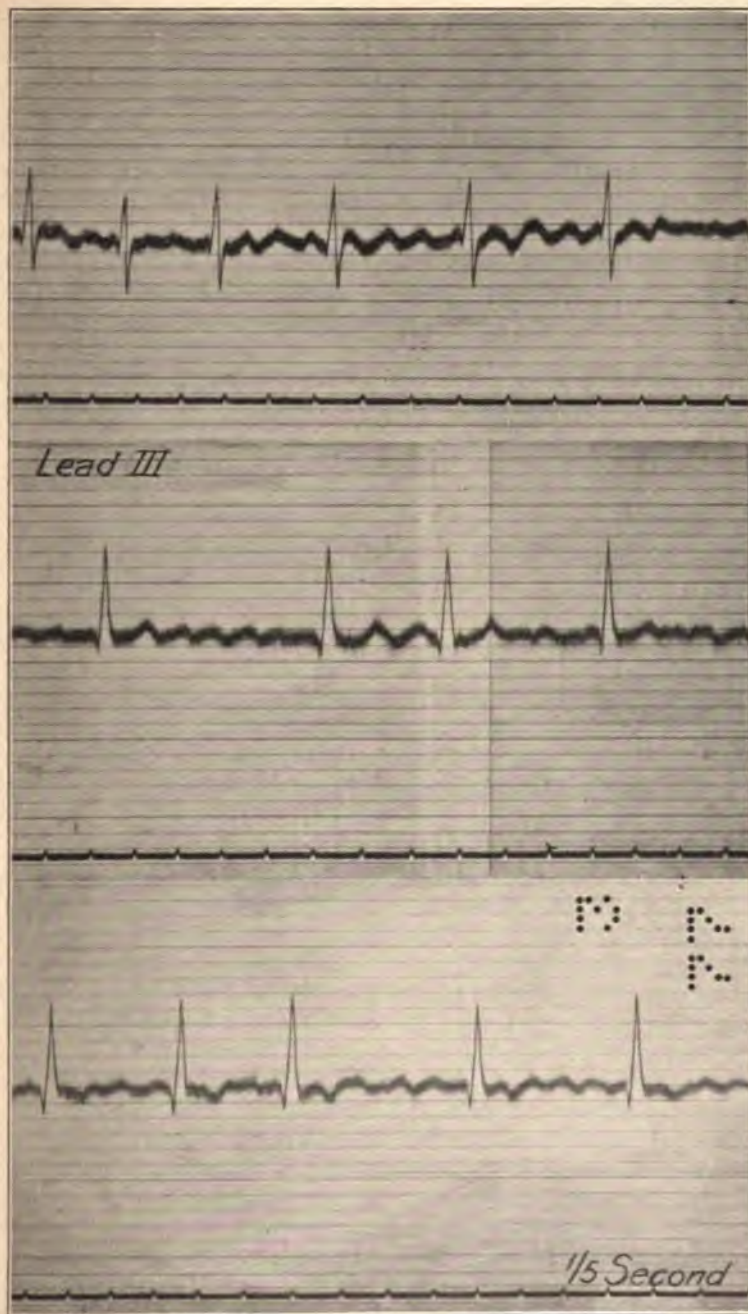
of the instruments depends upon the electrical changes that occur upon contraction of the heart muscle. The instrument records the action current of the heart muscle which, like all muscles, when it contracts, causes changes of electrical potential. The electrical impulses of the heart are conveyed from the patient by means of leads from various

FIG. 70



Normal electrocardiogram, showing variations in the several leads. Lead I, right arm and left arm. Lead II, right arm and left leg. Lead III, left arm and left leg. (James.)

FIG. 71



Electrocardiogram of a case of auricular fibrillation, showing irregularity of ventricular complexes in the distance apart and in height, and absence of summits P, which are replaced by irregular oscillations, differing in form and prominence. (James.)

PHYSICAL DIAGNOSIS

For ordinary clinical purposes the right arm and left leads are conveyed to the instrument by a pair of electrodes. Through these the impulses from the heart are transmitted to a very fine quartz thread suspended between two closely approximated electrodes.

This thread is deflected whenever a current of electricity passes through it. The movement is greatly magnified by a series of levers and is recorded on a photographic film by means of an arc light.

The normal electrocardiogram with a standard lead consists of two parts, an auricular and ventricular complex. The auricular complex is characterized by a summit or P wave. The ventricular complex consists of two upward summits, R and S, followed by a downward deviation, Q, and followed by a small upward deviation, S. These variations are characteristic of the normal heart.

The size and shape of the heart and changes in the position of the heart are determined by percussion. By percussion the area of relative cardiac dulness and the area of absolute cardiac dulness are determined.

Relative Cardiac Dulness.—This represents the area of the heart which is not covered by the lung and is unaffected by pulmonary conditions as a rule. The area of this area in normal adults extends transversely from the right border of the sternum to approximately 2 cm. of the midclavicular line on the left side. The upper border is at the level of the third rib, the lower border at the level of the fifth space just within the midclavicular line and is continuous with the liver dulness. The left border starts from the sternum about 3 cm. from the sternal margin and curves downward and outward toward the apex. Variations in this area are common. In children the heart lies more transversely and is proportionately larger, the apex usually lying in the fourth space. Changes in position of the heart, and the area of relative dulness will be correspondingly changed. For example, the apex falls toward the lower side when in the lateral decubitus, changing to the other side may make the apex move 3 to 5 c.c. to the one side or the other. On standing or lying down a somewhat similar change occurs. The apex is the most movable portion of the heart, the base least so.

The Area of Absolute Cardiac Dulness.—This corresponds with that portion of the heart not covered by the lung at the time of inspiration. The lungs overlap the heart, and in inspiration allow a small area to be in contact with the chest wall. The area is irregularly triangular in shape and extends from the fourth to the sixth costal cartilages. The right border may be roughly defined by a line drawn along the left edge of the sternum from the upper border of the fourth rib downward; the left border by a line extending from the upper

border of the fourth rib at the left edge of the sternum to a point midway between the sternal and the midclavicular lines in the fifth interspace. The lower border is continuous with liver dulness.

TECHNIC.—Of all the various methods devised for the percussion of the heart, probably the best method is by light immediate percussion. This may be performed by the ordinary method of percussion, *i. e.*, with the whole hand on the chest and the pleximeter finger in close apposition to the chest or by the orthopercussion method of Goldscheider. This method is particularly valuable in detecting the left border of the heart and the boundaries of the cardiohepatic angle. It is practised by using as the pleximeter the middle finger, bent at right angles, at the middle and terminal phalangeal joint, and placed parallel to the anteroposterior diameter of the chest wall. Only sufficient force is used with the plesser finger to bring out a note which can just be heard by the ear when in close proximity to the pleximeter finger.

The right border is determined by percussing from right to left toward the median line. Always begin to percuss sufficiently far from the heart to get the clear pulmonary note, and move toward the heart until the first impairment of the note is heard, which will correspond to the right border. The right border may correspond with a line outside of or along the right edge of the sternum (the normal), with the median line, or the left edge of the sternum, or even beyond the latter. When the edge of absolute dulness is reached, percuss with the finger parallel to the ribs to control the result previously secured; and as each interspace is percussed, the upper limit of liver dulness and the triangle (Ebstein's) between the liver and heart may be determined.

The left edge is determined by percussing in vertical lines from a point near the axilla toward the heart. The student should acquire the habit of proceeding from definite fixed positions toward the heart, and to observe the changes during inspiration and expiration. The upper border is determined by percussing from above downward, immediately to the left of the sternum. Over the upper sternum the great vessels impair the percussion note. The apex of the heart is difficult to outline and is usually determined by a combination of inspection, palpation, percussion, and auscultation. The lower border and rounded apex of an enlarged heart cannot be defined if the stomach contains food or fluid. When percussing the heart the various borders should be marked out on the skin with ink or a dermatographic pencil. In order to carefully watch any immediate change in the size of the heart and to preserve the present outline for future reference, measurements should be made of (1) the distance of the apex of the heart from the midsternal line, (2) the distance of the right border from the midsternum in the fourth space, (3) the distance from the aortic angle to the apex (the longitudinal diameter), and (4) the distance from the cardiohepatic angle to the upper left border (the transverse diameter). The first measure-

ment is normally about 9 cm., the second 4.3 cm., the longitudinal diameter 14.5 cm., and the transverse diameter 10.5 cm.

The *cardiohepatic* (or *Ebstein's*) *triangle* is the more or less resonant area in the right fifth interspace which separates the right heart from the liver. The apex of the triangle points to the right sternal edge, the base to the axilla. The upper side corresponds to the right border of the heart; the lower is the upper limit of the liver.

CHANGES IN SIZE.—Changes in size of the heart are best estimated by noticing an increase or decrease in the area of relative cardiac dulness, as this corresponds to the approximate size of the heart itself, while the area of absolute cardiac dulness, though more easily and accurately determined, is modified by intrapulmonary changes, and in reality outlines the margins of the lung in this situation.

Dulness Decreased or Absent.—It is diminished in emphysema. It is decreased or absent when the heart is drawn under the sternum by adhesions and when there is air in the pleural or pericardial sac, and at times as a result of extreme meteorism or dilatation of the stomach. Atrophy or absorption of pericardial fat in wasting diseases may cause an actual diminution in the size of the heart.

Dulness Increased.—Increase in the area of relative dulness in all directions occurs in hypertrophy and dilatation of the heart, and when the heart is pushed against the chest wall, as for instance by an aneurism or a mediastinal tumor, and when the patient leans forward. Increase in width of the dulness at the base of the heart occurs in dilatation, pericardial effusion, and aneurism of the aorta. Change in the position of the heart, a general idea of which is obtained by inspection and palpation, always changes the shape and extent of the dulness. The heart should be accurately delimited when displacement has taken place. Increase in the area of absolute dulness occurs in pericardial effusions and in flat-chested individuals.

Increase of Dulness Upward.—In pericardial effusion the area of dulness may extend upward, and this may be followed by extension of the right and left boundaries. The relative area of dulness becomes abolished; the change from pulmonary resonance to dulness becomes abrupt and decided; and the area of dulness becomes pyramidal or pyriform in shape. Upward increase of dulness may be due to disease of the vessels, especially of the aorta. Increase in the area of dulness over the bloodvessels is usually due to aneurism or dilatation. It may be general, as in dilatation of the aorta or fusiform aneurism of the aorta, or local, as in saccular aneurism. Extension of the dulness outward or upward from the normal line may be found at the right of the sternum (aneurism of the ascending aorta), or over the first bone of the sternum (aneurism of the transverse aorta), or to the left just above the cardiac area. In the last case the dulness is an extension upward of the normal area of cardiac dulness with rounding of the area affected, and is usually indicative of an aneurism situated at the beginning of the descending portion of the aorta.

Increase of Dulness to the Left.—This occurs in enlargement of the heart from hypertrophy or dilatation. If the dulness extends outward to the left and retains the triangular shape, with the apex pointed, it is due to hypertrophy of the left ventricle. If, on the other hand, it becomes quadrilateral in shape, with the apex rounded, it is due to dilatation of the left ventricle. Increase of dulness to the left upward signifies hypertrophy or dilatation of the left auricle. The oblique transverse diameter (the diameter from the cardiohepatic angle to the upper left border) is increased. In other cases increase in the dulness to the left occurs in displacement of the heart due to aortic aneurism or aneurism or mediastinal growth. In many of these cases the dulness due to the aneurism or tumor is continuous with that due to the heart, and an effort must be made to discriminate between them. The results of palpation and inspection aid in detecting the presence of one or the other of these conditions.

Obliteration of the Cardiohepatic Angle.—This takes place in pericardial effusion and dilatation of the right auricle.

Increase of Dulness to the Right.—This is due to hypertrophy and dilatation of the right auricle and ventricle. If the auricle is dilated, the right edge of dulness is extended beyond the normal in the third, fourth, and fifth, or as high as the second interspace. With this increase in dulness there is also seen, although not necessarily, because of the cardiac enlargement, an epigastric impulse.

Auscultation.—Method.—Either method of auscultation may be employed. By the immediate method we may form a general notion as to the condition of the heart sounds. The mediate, however, is preferable, because it is essential to localize the sounds that are heard, and because, if the double stethoscope is used, we can percuss the cardiac area. The patient should be in a comfortable position. The muscles should not be strained. The general directions for performing auscultation must be followed.

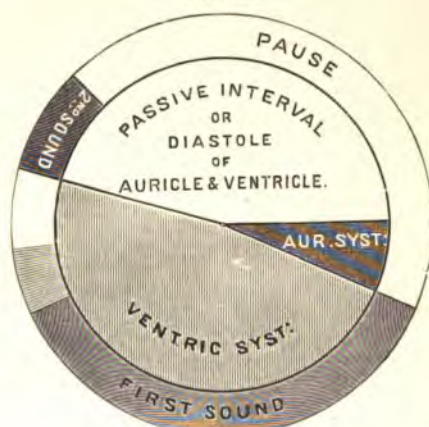
By auscultation we determine (1) the normal sounds of the heart, including their rhythm, their character, and the seat of the maximum intensity; (2) modifications of the normal sounds as regards (a) loudness and (b) rhythm; (3) the presence of abnormal sounds or murmurs.

The Normal Sounds.—The stethoscope is placed over the heart and the finger on the point of maximum impulse or the carotid pulse; a sound will be noted at the time of the ventricular beat or carotid pulse—the systole. This is followed almost immediately by another sound (occurring with the diastole) and then by a period of silence.

The sounds that attend the ventricular systole are known as the *systolic*, or first sounds. The sounds that follow are known as the *diastolic*, or second sounds. The sounds and silence mark the completion of a cardiac cycle, as far as the ear is concerned. A definite relationship in time exists in the cardiac cycle. *Cause:* The sounds created with the ventricular systole are due to contraction of the right ventricle and closure of the tricuspid valve under tension and to con-

traction of the left ventricle and closure of the mitral valve under tension. The rush of blood along the course of the vessels and the movement of the heart against the chest wall may contribute somewhat to the systolic sounds, but the first sound is largely muscular, with the added factor of the valves thrown into tension by the ventricular systole. The sounds heard in the beginning of the diastole (diastolic sounds) are due to closure of the aortic and the pulmonary valves. They are due to the tension produced on the valves as the respective arteries contract upon the columns of blood. The closure of the valves makes up most, if not all, of the diastolic sounds. In health the sounds of the

FIG. 72



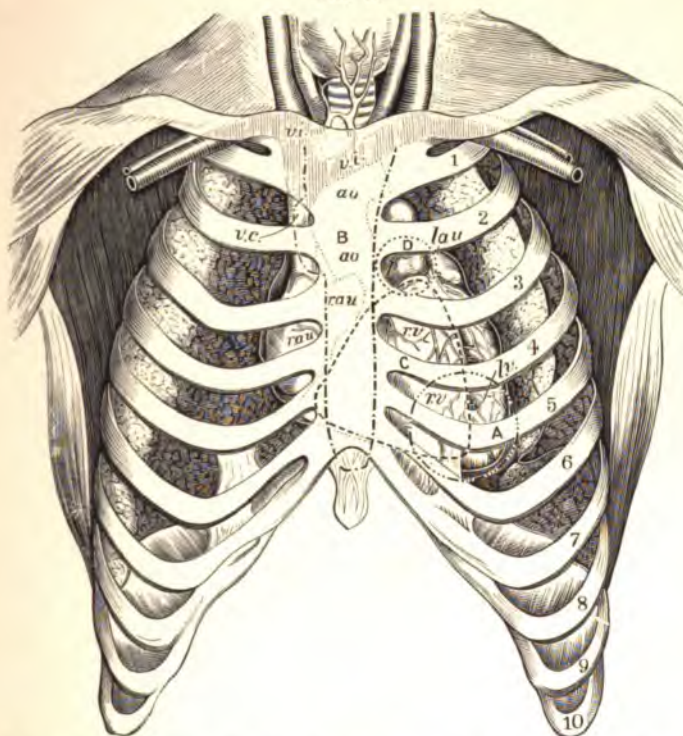
Diagrammatic representation of the movements and sounds of the heart. This diagram shows merely the general relations of the several events and does not represent exact measurements. In a heart beating seventy-two times a minute, Foster estimates each entire cardiac cycle as occupying about 0.8 second, of which 0.3 second represents the duration of the systole of the ventricle, 0.4 second the diastole of both auricle and ventricle, or the "passive interval," and 0.1 second the systole of the auricle. Only one "pause" is marked here—sometimes called the "long pause;" some writers describe a "short pause" also—indicated in the diagram by the small space between the first and the second sound.

systole blend because they are synchronous, and give the impression at a common point of one sound. The sounds of the diastole may or may not blend. Usually in health they do blend; often, however, there is an appreciable difference between the two, becoming more marked on altering the respiratory rhythm, as by a full breath.

RECOGNITION OF THE RESPECTIVE SOUNDS.—To distinguish the sounds we study their rhythm or time, their character, their position of maximum intensity, and their direction of transmission. We distinguish the first from the second sound by their rhythm and character, and then differentiate the elemental sounds of the systole and of the diastole by their points of maximum intensity.

The Rhythm or Time.—The sounds that are heard at the time of the normal cardiac impulse are the systolic or first sounds; the sounds that follow the impulse are the second sounds. The sounds that follow the long silence are the systolic or second sounds; those that precede the long silence are diastolic or second sounds.

FIG. 73



Areas of cardiac murmurs (Gairdner for the areas, and Luschka for the anatomy). The outlines of organs which are partially invisible in the dissection are indicated by very fine dotted lines; while the areas of propagation of valvular murmurs, as described in the text, have been roughly marked by additional much coarser dotted and interrupted lines—the character of the dots being different in each of the four areas. A capital letter marks each area, viz., *A*, the circle of mitral murmurs corresponding with the left apex; *B*, the irregular space indicating the ordinary limits of diffusion of aortic murmurs corresponding mainly with the whole sternum, and extending into the neck along the course of the arteries; *C*, the broad and somewhat diffused area occupied by tricuspid murmurs, and corresponding generally with the right ventricle; *D*, the circumscribed circular area over which pulmonic murmurs are commonly heard loudest. Reference letters: *r.a.u.* = right auricle; *a.o.* = arch of aorta; *v.i.* = the two innominate veins; *v.c.* = vena cava descendens; *p.* = pulmonary artery; *l.a.u.* = left auricle; *l.v.* = left ventricle; *r.v.* = right ventricle. (Finlayson.)

Character of the Sounds.—The systolic sounds are prolonged, somewhat dull in character, low in pitch, and resemble the sound produced pronouncing the syllable “ubb.” The diastolic sounds are short,

SOUNDS WEAKENED.—(a) *Causes outside of the pericardium.* (1) Thick chest walls; large mammary gland. (2) Emphysema of the lungs overlapping the heart. (b) *Affections of the pericardium*, as fluid or air in the pericardial sac. (c) *Conditions of the Heart.* Atrophy; some cases of dilatation; myocardial weakness from any cause, at times only.

MODIFICATIONS OF INDIVIDUAL SOUNDS.—The above applies to all the sounds. Increase or diminution of the systolic or of the diastolic sounds, or of any one of the four sounds, may be present.

Increase in Loudness of the Systolic Sound.—Increased loudness of the mitral first sound is noted when the muscle is hypertrophied and the tension on the valves thereby increased. The increase is most marked in hypertrophy (or dilatation) of the left ventricle. The sound is duller and has a prolongation which is very characteristic. In hypertrophy of the right ventricle the sound is dull and prolonged over the sternum, but not to the same degree as when the left is hypertrophied. In mitral stenosis the sound is sharp, slapping, and intense. Changes of any kind are difficult to recognize over the tricuspid area, while intensifying changes in the aortic and pulmonary first sounds are without much clinical significance.

Increase in Loudness and Accentuation of the Diastolic Sound.—Either of the second or diastolic sounds at the base may be accentuated. Cabot calls attention to the fact that normally up to the twentieth year of life the pulmonic second sound is more intense than the aortic, from then on the number showing such a change decrease, until after the sixtieth year practically every case shows an intensification or an actual accentuation of the aortic second.

1. *The Aortic Diastolic Sound.*—Anything that causes increased tension in the peripheral circulation will cause accentuation of the second sound. Arteriosclerosis and disease of the kidneys, particularly that form in which there are also general arterial changes, namely, chronic interstitial nephritis, are the usual causes, as they throw more work on the left ventricle and are usually associated with hypertrophy of the ventricle. An accentuation of the second sound is also associated with hypertrophy of the left ventricle; with atheroma of the aorta and aortic valves; and with aneurism or diffuse dilatation of the arch of the aorta. A transitory accentuation of the second sound, without pathological changes in the heart or artery, is observed in conditions of temporary increased blood-pressure, *e. g.*, during a chill or in an epileptic attack. The aortic second sound is intensified or louder than normal when there is overaction of the heart after muscular effort or excitement, or when any condition is present which intensifies all the sounds.

2. *The Pulmonary Diastolic Sound.*—Anything that heightens the tension in the pulmonary artery will cause accentuation of the pulmonary second sound. The causes are: (1) Any condition that produces marked congestion within the lungs, the right ventricle being at the

same time of normal or increased strength. There is accentuation in the early stages of pneumonia, and if the course of the disease continues favorable the sound may remain accentuated to the end. If the right heart is failing the sound will become fainter, and may be scarcely recognizable. Such change in the sound accompanies increase of respiratory distress, and indicates that the right heart is becoming exhausted. (2) Emphysema, phthisis, chronic bronchitis, and other chronic pulmonary conditions cause obstruction to the flow of blood through the lungs increasing the tension and consequently throwing more work on the right ventricle with resulting accentuation of the pulmonic sound. (3) Valvular disease of the heart affecting the mitral orifice (obstruction or insufficiency) which causes a backing up of the blood in the lungs and consequent increase of tension in the pulmonary artery. Sometimes in doubtful cases, either in the presence or in the absence of a murmur at the mitral orifice, the occurrence of this accentuation makes it more than probable that there is mitral valvulitis. All these chronic conditions cause increased amount of work upon the right ventricle, so that when there is an accentuated second pulmonic sound the correct inference is that the right ventricle is hypertrophied and functionally capable of performing its work.

Diminished Intensity or Feebleness of the Sounds.—(1) *Feebleness of the mitral sound*, observed at the apex of the heart, may be an indication of weakness of the muscle. It must be remembered, however, that weakness of the ventricle is not attended by enfeeblement of the sound alone, but that when the right or left ventricle is weakened, the duration of the sound is lessened. The loudness remains the same or may be increased. Note, then, that a short systolic sound, loud, sharp, flapping, and sometimes reverberating, heard at the apex, indicates myocardial weakness from any cause. The sound is caused largely by the contraction of the valves, the normal muscle sound being weakened. The systolic sounds become like the diastolic and may be differentiated by the ear with difficulty.

2. *Diminished intensity of the aortic second sound* is an indication of cardiac weakness and is apt to ensue in the course of fevers when exhaustion takes place. It is a sign of degeneration of the muscular walls of the heart. Under these circumstances the systole of the ventricle is also weakened.

Feebleness of the aortic second sound, with hypertrophy and hence strong contraction of the ventricle, occurs when the aortic leaflets are so dense that they are either unable properly to close on account of rigidity (aortic stenosis) or because they are so eroded that no valve sound is made (aortic insufficiency). In mitral stenosis and to a lesser degree in mitral insufficiency, the quantity of blood expelled by the ventricle is not sufficient to cause the valves to contract with accustomed sharpness. Similarly in shock and collapse the peripheral vessels are so relaxed that insufficient blood is thrown out and the aortic pressure is too low to cause the valve to close sharply.

3. *Diminished intensity of the pulmonary sound* is of importance in the course of valvular disease of the heart, providing previous accentuation has been observed. If the marked accentuation gives way to feebleness, there is strong probability that the right heart is undergoing dilatation with regurgitation at the tricuspid orifice. While accentuation of the pulmonary second sound in valvular disease is of good omen, enfeeblement of the sound is of bad prognostic omen, indicating weakness of the right ventricle.

ALTERATIONS IN THE RHYTHM OF THE HEART-SOUNDS.—*Fetal Rhythm of the Heart*.—Embryocardia is a term used to designate a condition of rapid heart action in which the pauses between the heart-sounds are of equal length. The first and second sounds are exactly alike, resembling the beat of the fetal heart. It is heard in all conditions which cause a pronounced rapidity of the heart (over 120), and hence is noted under conditions in which the heart is under an abnormal strain, as in fevers with high temperature, acute heart failure, and acute overwork of a chronically diseased heart, also in cases of paroxysmal tachycardia and allied conditions (Hirschfelder).

REDUPLICATION OF THE HEART-SOUNDS.—1. *Split Sounds*.—At times one of the two normal heart-sounds may be replaced by two sounds which are perfectly clear, of the same intensity and occurring very close together. Such a change is probably caused by a splitting of the sound into two parts and is due in all probability to asynchronism of the two ventricles, and in the case of the second sound, asynchronism of closure of the semilunar valves, with or without asynchronism of the ventricles. Split sounds are heard best at the base. A split first or second sound may be heard in any pathological condition which causes increased work on one or the other sides of the heart. In addition a split second sound may be due to any pathological condition which causes either increase of tension in the arterial circulation (chronic nephritis, arteriosclerosis, etc.), or in the pulmonary circuit (mitral stenosis, pulmonary diseases, etc.), resulting in asynchronism of closure of the two valves. Normally a split second sound may be heard after severe exercise or upon holding the breath as a result of increased intrapulmonary pressure.

2. *Gallop Rhythm*.—Reduplication with gallop rhythm, *i. e.*, the sounds resembling the hoof-beats of a galloping horse, is due to the introduction of an accessory sound in diastole so that three distinct sounds are heard, none of which are murmurs. The accessory sounds are heard best at the apex and are clear but less intense than the normal sounds. They may occur just before the first sound (presystolic), just after the second sound (protodiastolic), or in the middle of diastole (mesodiastolic). The accessory presystolic sound is probably due to the muscle sound produced by vigorous contraction of a hypertrophied auricle. The protodiastolic and mesodiastolic sounds are probably produced by the vigorous sudden slapping together of the auriculo-ventricular valves, before the actual beginning of systole, in hearts in

which there is present elasticity of the ventricular walls which distend, when filled quickly and early in diastole, and then elastically recoil to cause the sudden closure of the valves before the beginning of systole.

Clinically the presystolic gallop rhythm is produced in rapid, hypertrophied hearts working under a strain, as in pericardial effusion, exophthalmic goitre, chronic nephritis, arteriosclerosis, chronic cardiac disease, mitral stenosis, and acute febrile diseases. The other two accessory sounds are heard at times in aortic insufficiency or in dilatation of the heart when there is considerable residual blood in the ventricle, and they are frequently discovered upon auscultating slow hearts.

Abnormal or Adventitious Sounds.—Certain abnormal or adventitious sounds having a direct relationship to the cardiac cycle and caused by the action of the heart may be heard upon auscultation over the precordium or in its immediate neighborhood. Such sounds may be produced within the heart (endocardial) or may arise outside the heart (exocardial or pericardial). The endocardial adventitious sounds (murmurs) may be associated with pathological changes in the heart (organic murmurs), or may occur without any anatomical alteration in the heart (hemic, functional, or accidental murmurs).

The exocardial adventitious sounds may originate within the pericardium (friction-rub, splashing sounds); they may be the result of inflammation of the portion of the pleura in relation to the pericardium (pleuropericardial friction); they may originate in the portion of the lung in relation to the heart (cardiopulmonary murmurs), or they may be the result of mediastinal emphysema.

EXOCARDIAL ADVENTITIOUS SOUNDS.—*Intrapericardial Sounds.*—They are known as friction-rubs and splashing or bubbling sounds. The former occur in dry or fibrinous pericarditis and are due to the rubbing together of the roughened surfaces of the pericardium. The friction sound is recognized by (1) its character; (2) time; (3) position; (4) transmission; (5) movability; (6) modification by position of patient, pressure, course of disease, etc. The pericardial friction is usually of a to-and-fro character, and can be recognized as distinct from the heart-sounds. It resembles the rubbing or scraping together of two roughened surfaces; it is not necessarily synchronous with the normal heart-sounds. It is a to-and-fro sound, systolic and diastolic in time. It may, however, be only systolic or only diastolic; it is best heard over the area of absolute dulness; it is not transmitted away from the heart. Its location may shift from day to day in the precordial area; it may be modified by pressure or by respiratory movement or be influenced by the position of the patient. It may disappear entirely in the upright posture. An impression of nearness to the ear is given by the sound observed in the first stage of pericarditis. It may be increased or lessened in loudness by a deep inspiration. It disappears during the period of effusion, to return after that is absorbed.

Splashing sounds are heard when the pericardium contains air and fluid. There may be bubbling or gurgling, resembling the sound of a water-wheel. They are of the same rhythm as the heart.

Pericardial friction rub.—The sound produced by the rubbing of the heart against the pericardium, or *vice versa*, is heard over the right auricle and left ventricle and is increased by inspiration, decreased by expiration, synchronous with the heart-beat, but very frequently may be heard as a friction rub, synchronous with respiration. It is a dry, scratchy sound, and is of a friction rub in character.

Crackles.—At times a soft whispering vesicular sound may be heard either over the heart or over the lingula overlapping the heart (the lingula), at the left clavicle, or under the left scapula. These crackles are produced either by the impact of the heart on the alveoli and forcing out the air in them, or by the capillary matting or when a portion of the lung is compressed against the pericardium by adhesions, or else, by the retraction of the heart from a compressed position and its suddenly entering the alveoli. These murmurs are usually heard distinctly or only during inspiration, disappearing during expiration. They are usually systolic in time; they vary with the position of the patient; and they are modified by the position of the stethoscope. Such murmurs are without clinical significance and may be mistaken for endocardial murmurs by a careless observer. Occasionally nothing is heard but systolic or diastolic murmurs when bronchopneumonia is present in the lingula.

Crackles synchronous with the heart's rhythm.—When a mediastinal emphysema is present, a crackle synchronous with the heart's rhythm may be heard. It develops as a result of the pressure of the heart against the mediastinal vessels. The condition is extremely rare.

SOUNDS IN THE HEART.—MURMURS.—If the stethoscope is held over a large superficial vessel without pressure, no sound will be heard. However, pressure is employed, a sound or murmur is produced. The passage of the blood through the vessel produces no sound if the vessel or tube is of uniform caliber throughout. The pressure of the stethoscope alters the caliber and compels the fluid to pass through a narrow orifice into a wider space. In this manner a murmur is produced. The vibration of the molecules of the agitated fluid against the walls of the vessel into vibration and produces a murmur. The sound in this instance is carried in the direction of the blood currents, hence the murmur is known as an *onward murmur*. If the blood currents may take place. The fluid may flow backward from a narrower space without the production of sound; if, however, the fluid breaks on bevelled edges, as the leaflets of heart valves do, into the current, the fluid is again thrown into vibration and produces a murmur. If there is considerable constriction by the bevelled edges, the sound is carried farthest against the natural flow of the fluid—the *reverse backward murmur*. The character of a murmur depends upon the width of the orifice at which it is produced, upon the nature

of the walls of the orifice, upon the velocity and tension under which the fluid passes through it and upon the direction in which the flow occurs (Hirschfelder).

Murmurs are divided into two classes in accordance with their seat of development. Murmurs originating in the heart are known as cardiac murmurs. Murmurs originating in the bloodvessels are vascular murmurs. (See the Arteries.) Organic cardiac murmurs are always generated at the orifices from disease or from incompetency of the valves, or from a patulous non-valvular opening. The orifices are valvular and non-valvular.

MURMURS OF VALVULAR ORIFICES.—The valvular orifices and their anatomical relations have been described. Murmurs are produced at these orifices when they are open or when normally they should be closed. If the murmur is produced when the orifice is open, it is because there is narrowing of the orifice or dilatation of the cavity (relative narrowing). The murmur, then, is always produced with the natural current of blood, and hence is known as an onward or *obstructive* murmur. It always or nearly always implies organic disease at the valve orifice, accidental murmurs excluded. If the murmur is produced when the orifice should be closed, and hence when the valve leaks, it is because the valves are diseased and cannot shut the orifice, or because they are too small—incompetent—to shut it. Such murmurs are produced against the mitral current of blood, and are known as backward or *regurgitant* murmurs.

MURMURS AT NON-VALVULAR ORIFICES.—The orifices of the vena cava and of the pulmonary veins, and of the perforations of the septa in congenital heart disease, are non-valvular. They are at times the seat of murmurs, as in open foramen ovale or perforated ventricular septum. A patulous ductus arteriosus may also be the cause of a murmur.

DIAGNOSIS OF MURMURS.—It is necessary to determine, first, at which orifice the murmur is produced (the seat of the murmur), and second, the kind of murmur—obstructive or regurgitant. Murmurs are therefore studied as heart-sounds are studied, as to their position of maximum intensity, their time, and the direction of their transmission. The position of the murmurs indicates which valve orifice is affected. The time and the direction of transmission of the murmur indicate the nature of the lesion and the kind of murmur—whether obstructive or regurgitant.

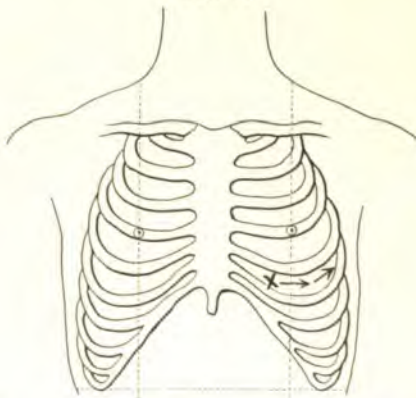
The Position of Maximum Intensity of the Murmur.—*The Orifice Affected.*—We are enabled accurately to determine the orifice at which the murmur is generated by noting the position of maximum intensity of the murmur. This position corresponds usually to the area at which the normal sound of the affected valve is heard loudest. It must be remembered that the cardiac orifices are closely situated, and that, therefore, the murmurs must be generated within a small area, so small that it would be impossible to ascertain at which valve orifice the

murmur is created, were it not for the fact that under the laws of conduction of sound the murmurs are conducted away from their point of origin to certain definite stations where in health the respective valve sound is also heard loudest.

1. *Murmurs at the Apex.—The Mitral Area.*—A murmur heard loudest or with the greatest intensity at the apex is known as a mitral murmur. It is created at the mitral orifice, but is conducted to the apex by the left ventricle, which is nearest the chest wall at this point. (See 1, Fig. 74, page 270.)

2. *Murmurs of the Xiphoid Cartilage—the Tricuspid Area.*—The murmur is heard loudest at the xiphoid cartilage or the head of the fourth or fifth rib. It is created at the tricuspid orifice, and is heard most distinctly over the lower portion of the sternum, and along the left edge, because the right ventricle is in apposition with the chest wall at this spot. (See 2, Fig. 74.)

FIG. 75



Maximum intensity of murmur of mitral regurgitation; systolic; transmitted to the left.

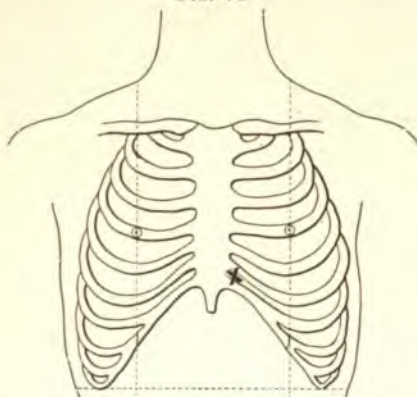
3. *Murmurs at the Second Costal Cartilage or Second Interspace on the Right—the Aortic Area.*—When a murmur is heard with greatest intensity at this point, it is usually generated at the aortic orifice, and is conducted to this region by the aorta, which comes nearest to the surface of the chest at this point. (See 3, Fig. 74.)

4. *Murmurs in the Second Left Interspace—the Pulmonic Area.*—A murmur heard loudest at the second interspace along the left edge of the sternum is usually generated at the pulmonary orifice; it is heard loudest in this area because the pulmonary artery is nearest the chest at this point. (See 4, Fig. 74.)

The Rhythm or Time of the Murmur.—The Kind of Murmur.—A murmur which is produced at orifices when they should be closed is known as the murmur of regurgitation, as the valve permits the blood to flow backward. A murmur that occurs when the blood should in

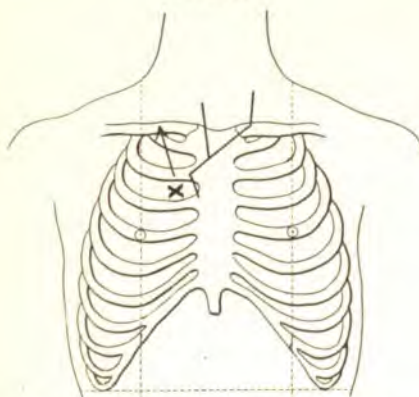
health be passing through an orifice is known as a murmur of obstruction, as the flow of blood is obstructed. We have to determine whether the murmur at an orifice is due to *regurgitation* or to *obstruction*. This is ascertained by the time of the murmur.

FIG. 76



Maximum intensity of murmur of tricuspid regurgitation; systolic.

FIG. 77



Position of maximum intensity and directions of transmission of murmur of aortic obstruction.

Murmurs with the Systole.—1. *In the Mitral Area.*—In health during systole the auriculoventricular valve is closed. The murmur indicates that there is such disease as to permit of a backward flow of blood or of regurgitation into the auricle. It is the murmur of *mitral regurgitation*. It may be due to disease of the valves (organic incompetency) or to relative incompetency. (See Fig. 75.)

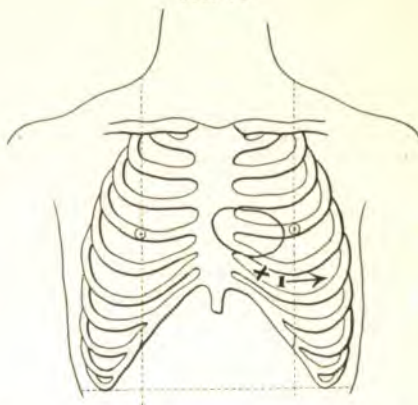
2. *In the Tricuspid Area.*—As on the left side, the murmur in this area is due to valvular disease, organic incompetency or relative

incompetency, which permits of regurgitation, *tricuspid regurgitation*. (See Fig. 76.)

3. *In the Aortic Area*.—During this time the blood is flowing from the ventricle into the aorta. If there is disease that causes obstruction at the orifice, the murmur of *aortic obstruction* is produced. The murmur may be due to disease or malposition of the aorta, or it may be an accidental murmur. (See Fig. 77.)

4. *In the Pulmonary Area*.—The pulmonary orifice is affected in the same way as the aortic orifice under the same circumstances. The murmur is due to pulmonary obstruction. It is exceedingly rare. A murmur here is more frequently accidental. (See Fig. 80.)

FIG. 78



Maximum intensity of murmur of mitral obstruction; presystolic, localized, or transmitted as area shows. 1, normal impulse; 0, area of reduplication of second sound.

Murmurs with the Diastole.—1. *In the Mitral Area*.—The blood is flowing from the left auricle to the left ventricle. Disease of the valves obstructs the flow. The murmur occurs in the beginning, in the middle, or at the end of the long silence. Mid-diastolic and late diastolic, or, because it occurs before the systole, presystolic are the terms applied to this murmur. It is the murmur of *mitral obstruction*. (See Fig. 78.)

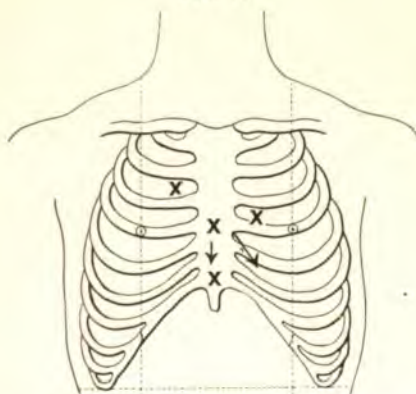
2. *In the Tricuspid Area*.—It occurs for the same reason and at the same time as the diastolic murmurs generated at the mitral orifice. It is rare; although it is more common to find tricuspid obstruction than is usually supposed.

3. *In the Aortic Area*.—The aortic valve closes in the diastole. A murmur indicates that it is so diseased that it cannot prevent blood flowing backward or regurgitating into the ventricle. It is the murmur of aortic regurgitation. A murmur of the same time and in the same situation may be due to dilatation or aneurism of the aorta. (See X, Fig. 79.)

4. *In the Pulmonary Area.*—A diastolic murmur in this area is due to regurgitation at the pulmonary orifice. (See Fig. 80.)

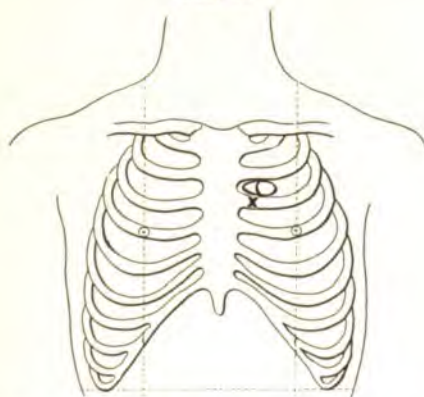
The Direction of Transmission.—It depends upon the situation of the murmur and the time at which it is produced. Some murmurs are not transmitted. The transmission is usually in the direction of the currents that produce them.

FIG. 79



Positions of maximum intensity and directions of transmission of murmur of aortic regurgitation.

FIG. 80



X, maximum intensity of pulmonary diastolic murmur; O, area of murmur of anemia.

Murmurs on the Mitral Area.—To the Axilla.—A murmur heard at the apex with systole and caused by regurgitation at the mitral orifice, is transmitted into the axilla and may be heard at the angle of the scapula. The murmur produced in the same area before the systole—obstruction—is usually not transmitted. It is heard at the apex, or

valves, the blood is forced back to the point of maximum closure, and is driven forward. Sometimes it is transmitted to the walls of the large vessels. (See Fig. 13, p. 283.)

Diastolic murmurs.—These are the murmurs of regurgitation, or backflow. They are usually heard (1) during the diastole, and (2) at the apex of the heart.

Murmurs of aortic regurgitation.—These are heard in the fourth or fifth intercostal space, at the base of the heart, and are transmitted in the direction of the regurgitant flow. They are usually heard by the stethoscope and by the hand, and are usually accompanied by a thrill at the base and in the axilla.

Murmurs of mitral regurgitation.—These are heard in the fifth intercostal space, at the apex of the heart, and are transmitted in the direction of the regurgitant flow. They are usually heard by the stethoscope and by the hand, and are usually accompanied by a thrill at the apex and in the axilla.

Murmurs of aortic stenosis.—These are heard in the second or third intercostal space, at the base of the heart, and are transmitted in the direction of the regurgitant flow. They are usually heard by the stethoscope and by the hand, and are usually accompanied by a thrill at the base and in the axilla.

Murmurs of mitral stenosis.—These are heard in the fifth intercostal space, at the apex of the heart, and are transmitted in the direction of the regurgitant flow. They are usually heard by the stethoscope and by the hand, and are usually accompanied by a thrill at the apex and in the axilla.

The character of the murmur.—The murmur may be soft or blowing, or may be harsh, or may be whistling. They may be high or low in pitch. Murmurs that are soft and high in pitch are usually due to disease of the valve, or to a narrowing of the orifice, or to the presence of a stenotic part in the lumen of the artery. Murmurs that are soft and low in pitch are usually due to a regurgitant condition which causes swelling of the valve or venous or soft excitation, they are heard in endocarditis of the valve or in the malignant form of endocarditis. The only murmur that has special characteristics is the murmur of aortic stenosis. It is a prolonged crescendo murmur of a whistling or grating character, sometimes rising as if fluid were being forced through a narrow channel. It is usually presystolic, but may occur elsewhere in diastole.

Intensity.—The loudness of the murmur is not of special significance although, in general, it may be said that it indicates good compensation, and that the heart muscle is sufficiently strong to meet the demands

of the circulation. Murmurs are louder in the recumbent than in the erect posture in some instances, especially mitral and tricuspid murmurs. Murmurs are often more distinct after exertion. Loud murmurs may become weak, and this change in the character of the sound is of serious omen. They may disappear in the course of fevers and during the terminal state.

Disappearance of Murmur.—This is probably due to the fact that there is complete compensation. In the terminal stages of cardiac disease they disappear because of weakness of the heart muscle. Rarely they disappear because the roughened valve causing them has been repaired. In other cases it may be necessary to bring out a faint murmur or increase its intensity by having the patient move about; this renders it most distinct by inducing more rapid action of the heart.

The Significance of Murmurs.—Murmurs other than accidental heard at the various orifices indicate either (1) disease of the valves; (2) relative incompetency of the valves; or (3) diseases of the vessels in intimate relation with the heart. The systolic murmur at the second costal cartilage on the right may be heard when there is disease at the aortic orifice causing obstruction; in atheroma of the aorta; in cases of aneurism just above the valves. Before concluding that the murmur is due to disease of the valves, we must be able to exclude the other conditions.

Atheroma of the aorta in old people is most difficult to distinguish from obstruction because the character of the murmur is the same and the associated conditions are similar. In both there may be a previous history of syphilis, gout, or alcoholism. These diseases are associated with atheroma in other arteries of the body, and with the degenerative changes that accompany atheroma. In young persons with a direct history of rheumatism or when the process has followed septicemia, the probabilities are in nearly all cases that the murmur is due to aortic obstruction, but in subjects under forty, lacking such a history, the probability is that the murmur is produced by syphilitic aortitis, which is very frequently associated with aortic insufficiency.

In other valve areas the chief task is to decide whether the murmur is organic, due to valvulitis or relative incompetency of the valve, or accidental (inorganic).

Murmurs Due to Relative Incompetency.—The valves are sometimes unable to close properly if the cavity of the ventricles increase in size, stretching the valvular sphincter so that the valves do not coaptate to close the widened orifice. The tricuspid and mitral valve leaflets often become thus incompetent. Mitral and tricuspid regurgitation ensue. The murmurs are soft and low in pitch and not widely transmitted; the heart is dilated.

ACCIDENTAL, HEMIC, FUNCTIONAL, OR INORGANIC MURMURS.—These murmurs occurring without valvular lesion are called "accidental" because of their evanescent character; "hemic" or "anemic" because

They are derived from the heart and the pulse. They depend upon the secondary effect of the lesion upon the heart and upon the circulation.

In *aortic obstruction*, on account of obstruction to the flow of blood, the left ventricle hypertrophies; moreover, the blood-stream is lessened in volume, the pulse is small and of high tension. The physical signs of hypertrophy of the left ventricle and a small slow pulse are corroborative evidence of this lesion at the aortic orifice.

In *aortic regurgitation* the blood flows back into the ventricle. On this account, therefore, some dilatation takes place, a dilatation which, if compensation is perfect, is overcome by hypertrophy. The signs of enlarged left heart are present, however, as shown by inspection, palpation, and percussion. But the pulse of aortic regurgitation is of the greatest diagnostic significance. With the finger on the radial the impression is at once received of recedence of the pulse wave as soon as it strikes the finger. This is more marked if the hand is elevated. It is the water-hammer or Corrigan's pulse.

In *mitral regurgitation* the left auricle does not change, but the stress is thrown upon the right side of the heart, and we have the signs of right-sided hypertrophy and dilatation; but more marked than this is the evidence of high tension of the pulmonary artery, shown by accentuation of the second sound. In mitral regurgitation the blood flows back into the auricle and when the right heart weakens engorges the venous system. The arterial system is in consequence devoid of blood, hence the arteries are empty.

In *mitral obstruction*, in addition to the characteristic murmur, the thrill is of great significance. Moreover, the left auricle hypertrophies and shortly afterward the right heart. The pulmonary second sound is accentuated and frequently reduplicated. The pulse is small and feeble.

MULTIPLE CARDIAC MURMURS.—More than one murmur may be heard over the heart. The murmur depends upon the number of valves that are the seat of disease and the lesions at the orifices. We may have valvulitis of the aortic, mitral, and tricuspid valves conjoined. More commonly one valve is diseased, giving rise to a murmur, while another valve is relatively incompetent, on account of dilatation of one or more of the cavities of the heart, and a murmur is thus generated at its orifice. It is common to see aortic obstruction from valvulitis and mitral regurgitation from incompetency; mitral obstruction from valvulitis and tricuspid regurgitation from incompetency. The diagnosis of the various murmurs will be discussed in the section on Valvulitis.

The Arteries.—The stethoscope should always be used in examining the arteries. The double stethoscope is preferable, as strong pressure must be avoided upon the vessels. The arteries open to auscultation are the carotid, when the head is slightly extended; the subclavian; the innominate, above the sternoclavicular articulation; the brachial artery, in the bend of the elbow; and the femoral artery, just below Poupart's

ligament. The normal systolic and diastolic heart sounds are often heard in the carotid and subclavian arteries. The systolic sounds may be heard over the abdominal aorta, due to tension of the vessels. The diastolic sound is rarely heard in this situation. In other vessels no sounds are heard.

Induced or Pressure Murmur.—By pressure with the stethoscope over one of the vessels its caliber is modified and a murmur created. This murmur corresponds in time with the pulse, hence it is systolic, and increases or diminishes in intensity, depending upon the amount of pressure placed upon it. Here may be mentioned also the systolic humming which is heard in children between the third month and the sixth year over the fontanelles and over the gravid uterus in women.

Diseases outside of the bloodvessels may give rise to what may also be called pressure murmurs. When heard over the subclavian artery the pressure murmur may be due to adhesions or solidifications at the apex of the lung. It is more frequently heard at the left, and may only be present during full expansion of the lung. It is due to temporary pulling or bending of the artery during deep breathing. When it occurs on both sides, it is not of much significance. Murmurs in the axillary artery, or in any arteries surrounded by enlarged lymphatic glands, are created by their pressure. Murmurs in the thyroid gland have been referred to. (See Goitre.)

Abnormal Murmurs.—Abnormal sounds or murmurs are due to diseases outside of the vessels causing pressure, and disease of the vessels. Murmurs from disease of the vessels, as the aorta, are discussed under the head of arteriosclerosis or aneurism.

Conduction Murmurs.—Murmurs may be propagated into the arteries. A systolic murmur created at the aortic orifice may be heard in the vessels of the neck and along the aorta. On the other hand, in aortic regurgitation the diastolic sound normal in the carotid and subclavian disappears, and the diastolic murmur is not heard.

Double Sounds of the Vessels.—Double sounds are sometimes heard in the femoral artery under the following circumstances: (1) in aortic insufficiency; (2) in mitral stenosis; (3) in lead poisoning; (4) in pregnancy; (5) in aneurism. Duroziez's double murmur, heard when sufficient pressure is used by the stethoscope, occurs in aortic regurgitation when there is good compensation. Many authorities refer to this as a valuable diagnostic sign in this affection. It is probably caused by sudden collapse of the artery and the reflux of the blood current.

Murmurs in Relaxed Vessels.—The increased caliber favors the development of a murmur by the creation of a fluid vein. Dilatation of the innominate artery sometimes takes place, giving rise to a murmur, which in loudness and character simulates the murmur of aneurism.

Murmurs Due to Disease of the Arteries.—In the aorta the murmurs are due to aneurism or atheroma, or both. They may be systolic or diastolic. In the smaller vessels both conditions may be present, although atheroma is the usual one. The murmur is systolic in time,

rough in character, strong or weak. It is associated with other signs of atheroma.

The Veins.—In health no sounds are heard. Two conditions contribute to the creation of a murmur in the veins: (1) change in the character of the blood; (2) dilatation with occurrence of positive venous pulse.

The Venous Hum.—In anemia and chlorosis, and sometimes in health, a hum or murmur or buzzing sound is heard over the jugular veins. It is louder on the right side than on the left. It is soft and low in pitch, and may be musical; it is described as a continuous humming or whizzing. For its detection a double stethoscope should be used, as pressure increases it, and the patient should not turn the head to one side, as it is increased when this position is taken. The murmur is modified by the respiration and by the cardiac action. It is louder in deep inspiration, when the blood is going more rapidly to the thorax. It is also louder in the upright position. It is frequently louder during the diastole. The increased loudness at these periods occurs because, from the sucking action during inspiration and during the diastole, the blood is drawn more rapidly toward the heart. The murmur is caused by the flow of blood from the narrow jugular into its wider bulb with greater rapidity than normal, owing to the change in the blood in anemia. The venous hum may also be heard in the innominate veins (first and second interspaces and right costoclavicular articulation) and in the subclavian and axillary veins. A venous hum is sometimes heard at the lower border of the liver, to the right of the median line or over the xiphoid cartilage in cirrhosis of the liver. It is created in the enlarged collateral veins. It may be modified by pressure of the stethoscope. It may be heard in this situation in emaciated and cachectic individuals not the subject of cirrhosis.

CHAPTER XXI

PHYSICAL DIAGNOSIS OF DISEASES OF THE LUNG

Physiology.—Respiration is a reflex act in response to a stimulus received in a nervous centre and transmitted by efferent fibers to the muscles of the throat. The expansion of the chest causes air to fill the alveoli of the lungs and between them and the pulmonary capillaries the respiratory exchange of gases take place. The excitation for *inspiration* is supplied by the carbon dioxide in the circulating blood and by stimulus derived from collapse of the lung. The impulses are conveyed by afferent nerve fibers to the inspiratory centre in the medulla, which is connected by efferent fibers with the muscles of inspiration. *Expiration* is largely a passive act. The presence of oxygen in the blood and the expansion of the lung excite the rhythmic action of the expiratory or inhibitory centre in the medulla, controlling the expiratory muscles in forced expiration.

The Value of Physical Diagnosis.—By physical examination of the lungs (1) the degree of activity (movement) and (2) the physical condition of the parts subjected to examination are ascertained, but the disease itself is not diagnosticated. Abnormal signs simply indicate an abnormal condition which may be due to any number of diseases. As the lungs in health contain air, any physical change that takes place in them causes either an increase or a diminution in the amount of air they contain. This may be general (bilateral), or it may be limited to one side (unilateral), or to a small area (local). An increase or diminution in the amount of air in a part suspected to be the seat of disease is determined by percussion. As adjuncts are the phenomena that can be elicited by means of inspection, palpation, and auscultation, which depend upon the movements of the lungs and upon the sounds produced in breathing and speaking.

Topographical Anatomy.—In order that the condition of the underlying parts may be learned from the physical signs elicited upon the chest, a knowledge is required of the relations of the various organs with the chest wall.

Lungs.—The apices of the lungs extend in front 3 to 7 cm. (one and one-fifth to two and three-quarter inches) above the clavicles, the right apex being about one-half inch higher than the left; behind, to a line drawn transversely through the spinous process of the seventh cervical vertebra. The lower margin of the right lung, when the chest is passive, commences in front at the insertion of the sixth costal cartilage into the sternum, runs parallel with the upper border of the sixth rib to the axillary line, and then descends to the upper margin of the seventh

PLATE IV

FIG 1.—Anterior Aspect.

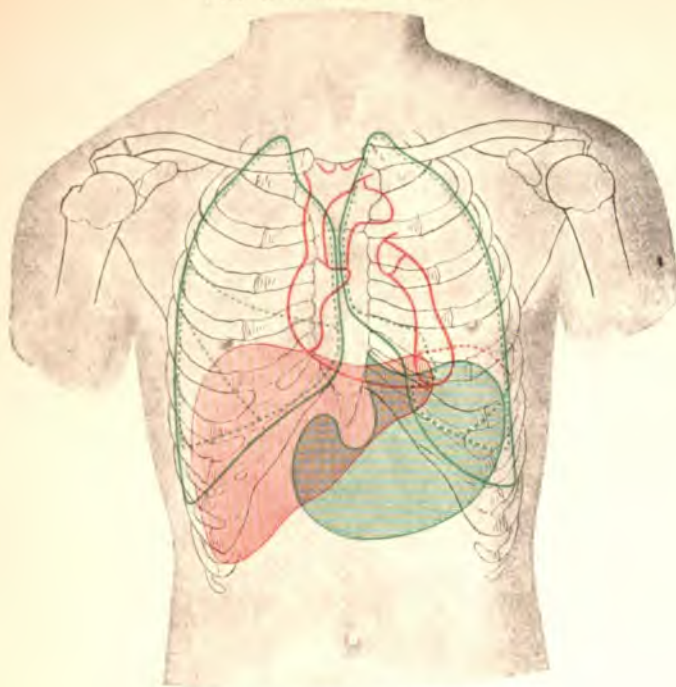
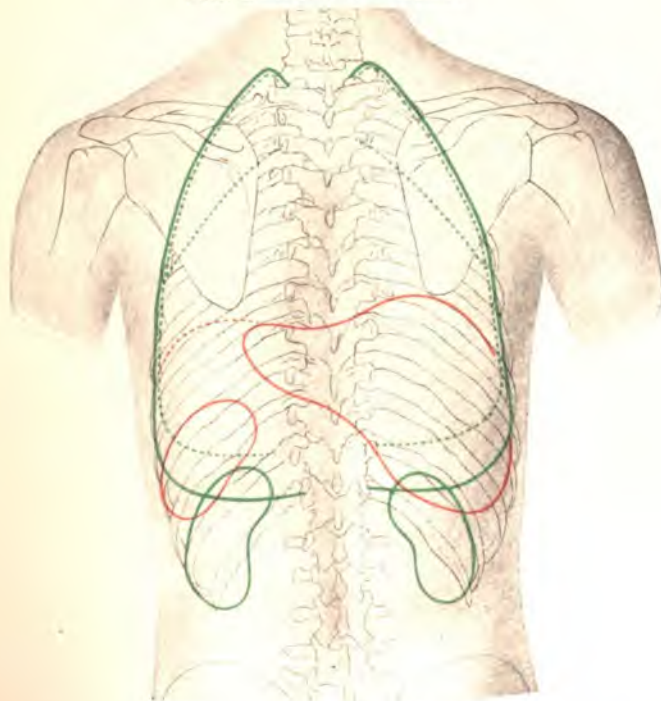


FIG. 2 —Posterior Aspect.



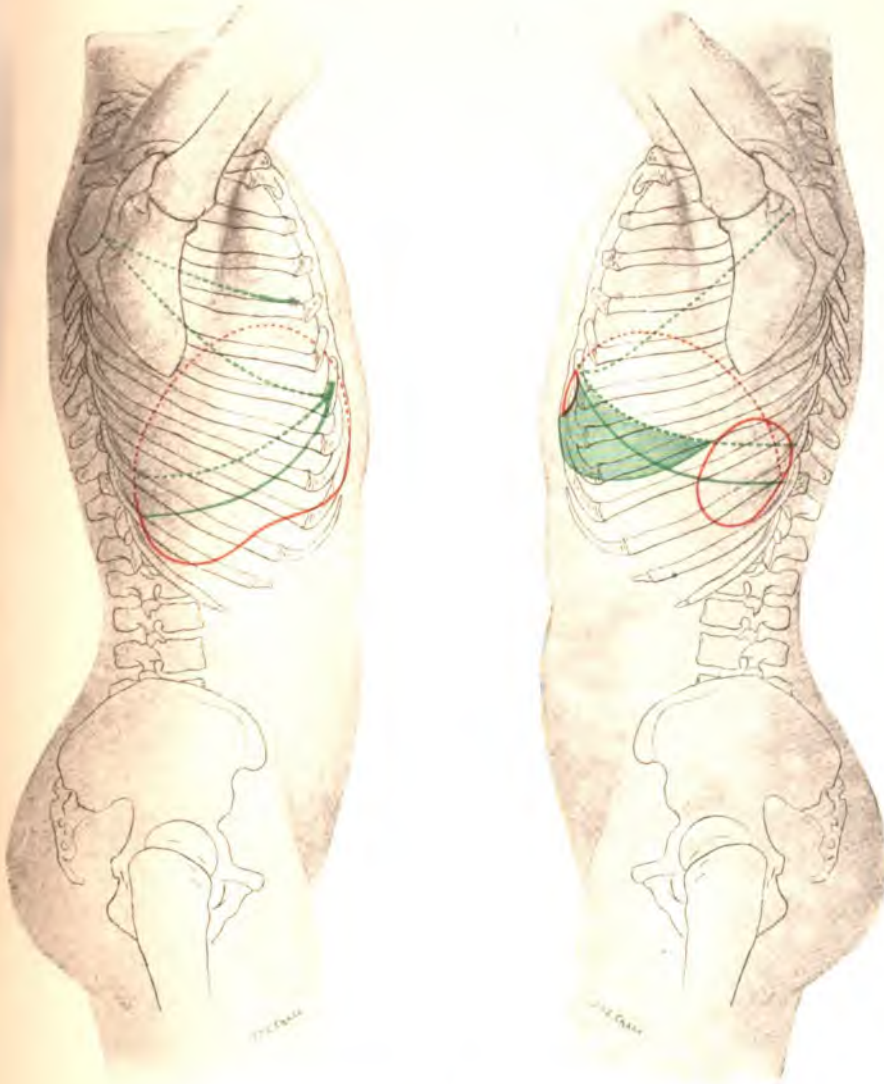
Situation of the Viscera.

—broad red lines—Margins of heart and vessels—broad red lines. Margins of lungs and individual
 —dotted green lines. Limits of pleural sacs—solid green lines.
 —green shading—Stomach—green shading.

PLATE V

FIG. 1.—Right Lateral Aspect.

FIG. 2.—Left Lateral Aspect.



Situation of the Viscera.

Margins of lungs and of individual lobes—dotted green lines. Limits of pleural sacs—solid green lines. Liver and spleen—solid red lines. Diaphragm—dotted red lines. Stomach (portion not covered by lung)—green shading.

rib. The lower margin of the left lung begins at the sixth costal cartilage. Posteriorly the lower border of both lungs extends to the tenth rib. With full inspiration the lungs descend both in front and behind almost the extent of one interspace.

In the axillary lines the extreme position of the lung border may reach 4 cm. above and below the mean, so that the total excursion may be 8 cm. with deep respiration. Change from the dorsal decubitus to the erect posture will usually raise the lung and liver boundary owing to increased tension of the abdominal walls. Change from the dorsal to the lateral decubitus will lower the lower pulmonary boundary of the upper lung in both mean position and full inspiration about 4 cm., so that the total excursion of the lower boundary is increased to perhaps 10 cm.

The position of the lungs and of their lobes is shown in Plates IV and V. The *upper lobe* of the *right lung* extends in front downward to the fourth rib, laterally to the third, and behind to the spine of the scapula. The *lower lobe* extends to the tenth rib behind, and when fully expanded, in the axillary region as well. The *middle lobe* is not seen behind; in the axillary region it extends from the third to the fourth rib in inspiration; in front, from the lower margin of the upper lobe to the sixth rib. The *upper lobe* of the *left lung* extends to the sixth rib in front, to the fourth interspace at the side, and a little above the spine of the scapula behind. The *lower lobe* extends to the base of the lung behind and to the level of the eighth rib at the side.

Pleuræ.—The pleural sacs extend below the lower border of the lungs; two inches in the midclavicular line, three and one half inches in the midaxillary line, and one and one-half inches in the scapular.

The Landmarks of the Chest.—For the purpose of bearing in mind the relations of the organs to the surface of the chest, and the localizing and proper recording of the seat of the disease, certain landmarks are employed, consisting of anatomical points on the surface of the chest and artificial vertical lines.

The Imaginary Vertical Lines.—Of these, there are six drawn to the right and to the left of the median line: (1) the *sternal line*—the lateral border of the sternum; (2) the *midclavicular line*, drawn from the middle of the clavicle, generally passing through the nipple in males; (3) the *anterior axillary line*, drawn from the anterior fold of the axilla; (4) the *midaxillary line*, drawn from the centre of the axilla; (5) the *posterior axillary line*, drawn from the posterior fold of the axilla; (6) the *scapular line*, drawn through the angle of the scapula when the arm is at rest at the side of the patient.

The Ribs and Interspaces.—These are used as transverse lines. The *first rib* corresponds to the clavicle; the first interspace is the region between the clavicle, or first rib, and the second rib; the number of interspace corresponds to the number of the rib above it. It is important to remember, particularly when *counting the ribs of fat* persons that the finger passed down from the top of the sternum soon

FIG. 81

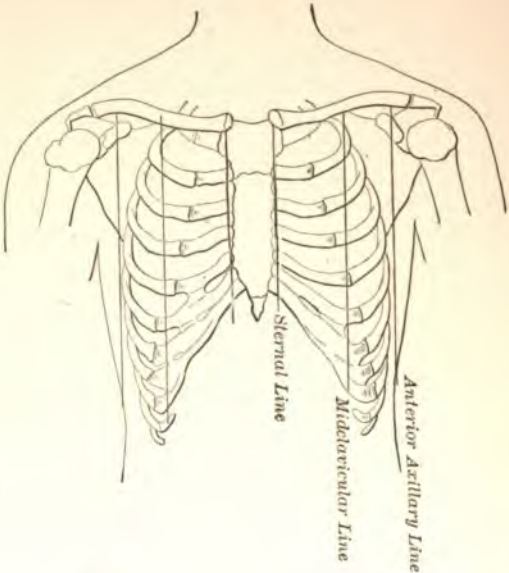


FIG. 82

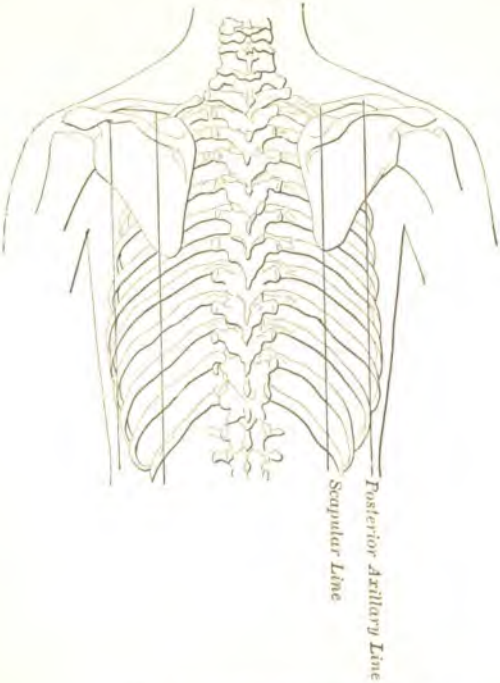
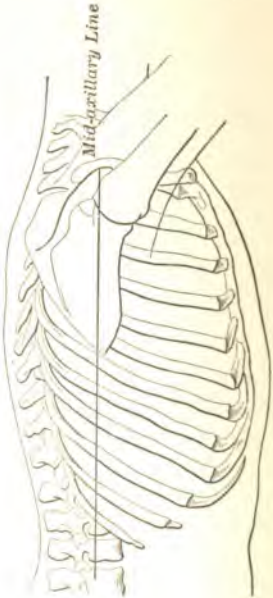


FIG. 83



Imaginary lines for purposes of localization and recording.

comes to a transverse projection, slight, but always to be felt, at the junction of the first with the second bone of the sternum which corresponds with the middle of the cartilage of the second rib; that the scapula lies on the ribs from the second to the seventh, inclusive; that the eleventh and twelfth ribs can be felt even in corpulent persons. outside the erector spinæ, sloping downward. It is important to recognize the *relation of the ribs to the vertebræ*. The first rib articulates with the first dorsal vertebra, which can be located by the position of the prominent spine of the seventh cervical vertebra; even in very fat people this prominence can be recognized. The remaining ribs, except the tenth, eleventh, and twelfth, have facets of articulation on two vertebræ; as the second rib with the first and second thoracic vertebræ. The eleventh and twelfth articulate with the eleventh and twelfth thoracic vertebræ.

The Angles of the Thorax.—The *costal angle* is the angle the costal cartilage makes with the sternum. It varies during the act of respiration. In inspiration the rib rises, as the sternum projects, and apparently elongates, the angle becoming more obtuse; in expiration as the sternum falls the rib becomes more slanting and the angle more acute.

The *epigastric angle* is formed by the meeting of the costal margins of both sides at the xiphoid cartilage. On inspiration it is obtuse, increasing as the ribs rise; in expiration it is more acute. The junction of the first and second portions of the sternum is known as the *angle of Ludwig*. It is opposite the middle of the second rib, and is on a plane with the lower border of the fourth dorsal vertebra.

Other Topographical Landmarks.—The *top of the sternum* is on a plane with the lower border of the second dorsal vertebra. The *junction of the body of the sternum with the xiphoid cartilage* is on a plane with the lower border of the eighth dorsal vertebra.

THE REGIONS OF THE CHEST.—In the anterior portions of the chest the regions are: the *supraclavicular region*, above the clavicle; the *infraclavicular region*, extending from the clavicle to the third rib; the *mammary region*, between the third and the sixth ribs. Laterally there are two regions, the *axillary* and the *infra-axillary*, separated by the sixth rib. Posteriorly the regions are the *suprascapular*, the *scapular*, the *infrascapular*, and the *interscapular*, the last being the region between the scapulæ.

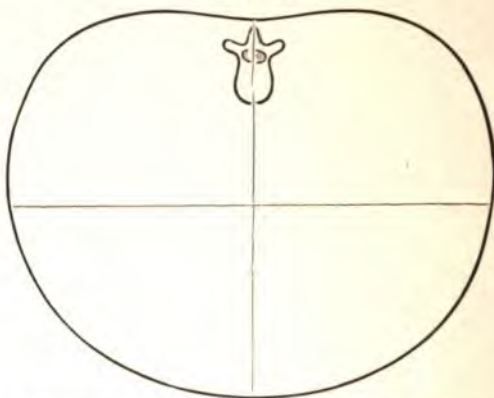
ABNORMAL BOUNDARIES OF THE LUNG.—The lungs are increased in size in emphysema. Diminution in size, shrinkage, or contraction occurs at the apices or along the lateral borders of the lower edges in phthisis or in retracting pleurisy. It may be general in atelectasis. The size of the lung apparently varies in diseases below the diaphragm, as when an enlarged liver or ascites compresses the pulmonary tissue.

Inspection of the Chest.—By inspection of the chest we learn (1) the appearance of the external surface, (2) the shape and size, and (3) the movements.

The Appearance of the External Surface of the Chest varies in health and disease. The external veins over the surface of the chest and capillaries along the base are enlarged and more distinct than normal when there is an intrathoracic obstruction as a tumor, or aneurism. The veins, together with the cervical veins, are also enlarged in dilations of the right heart. In cancer of the breasts and during lactation the veins around the breast are prominent. *Edema* or *subcutaneous emphysema* may be noted as the result of conditions mentioned under General Inspection.

Asymmetrical prominence of the ribs and wasting of the fat and muscles is seen in muscular atrophy or paralysis. Similar changes occur symmetrically in wasting diseases as cancer, diabetes, etc. Phthisis causes a localized wasting as seen in the clavicular and suprascapular muscles before wasting has become general.

FIG. 84



Transverse section of healthy male adult chest. Semicircumference, right side, $16\frac{3}{4}$ inches; left side, $16\frac{1}{2}$ inches; expansion, $3\frac{1}{2}$ inches. (Ward 6, Philadelphia Hospital.)

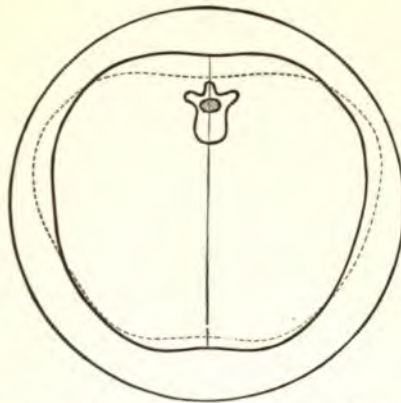
The Shape and Size of the Chest.—In health the shape of the chest in a cross-section is that of an ellipse, described as reniform. The anteroposterior diameter is about one-fourth less than the transverse diameter. In children a cross-section is more circular. The chest should be symmetrical, the right side probably a little larger than the left. In *disease* the chest may be (1) enlarged or (2) diminished in size. Such changes may be (a) general or bilateral (b) unilateral (c) local. The element of time is necessary to produce bilateral enlargement or diminution in size of the chest and unilateral diminution. Unilateral enlargement and localized changes are usually rapid in development.

GENERAL OR BILATERAL CHANGES IN SHAPE.—These may be (I) disease or to (II) developmental faults.

I. 1. *The Emphysematous Chest.*—The chest is reniform shaped, all the diameters are increased, particularly

The ribs are elevated, almost horizontal, making the epigastric angle obtuse. The spine and the sternum are arched; the shoulders rounded and elevated; the scapulæ lie flat against the thorax, and the muscles of respiration stand out prominently. Bilateral pleural effusions and rapidly growing cancers of the lung are also said to cause a chest of this form.

FIG. 85



Bilateral enlargement of emphysema. Inner line = emphysematous chest; outer line = a circle drawn to show how nearly the emphysematous approaches the circular shape; dotted line = normal adult chest. Measurements of normal chest. Circumference = 89; transverse = 29.6; anteroposterior = 22.25; of emphysematous chest, 87.75, 27.25, 25.4. (Gee.)

2. *The Rachitic Chest.*—In rachitis the chest wall is extremely flexible as a result of changes in the bones. This alone may cause deformities in shape but there is usually added a hyperplasia of the lymphoid structure of the pharynx, causing an obstruction to respiration which tends to exaggerate the skeletal changes. On the other hand respiratory obstruction may cause changes in shape similar to those that occur in rickets without the disease being actually present. Various types of rachitic chests may be seen, but two particular types are most characteristic. In the first type there is found a sinking-in of the sternum, and a vertical depression at the osteocartilaginous articulations of the ribs, which in turn are swollen and beaded, forming the *rachitic rosary* which usually disappears in later childhood. In the second type the sternum is prominent, the lower portion projecting to an unusual degree; the chest is shortened, and instead of the square front of the first there is a narrow keel-shaped projection suggesting the name applied to the condition, "pigeon breast." The costal angle is very acute and a rosary is also present. Simple rachitis results in a chest of the first form; respiratory obstructions may cause the second form, but rachitic children are more susceptible to changes in intrathoracic than normal children and hence the condition is usually a form of rachitic chest.

3. *The Diseased Vertebra Chest.*—Disease of the thoracic vertebræ with abnormal curving of the spinal column usually results in a shortening of the chest with prominence or sinking of the sternum, although many other deformities may occur. Kyphosis will usually cause symmetrical, scoliosis, asymmetrical changes in the shape of the chest.

FIG. 86

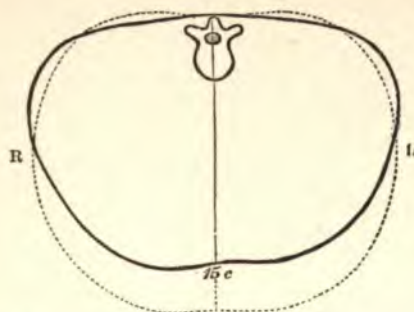


Emphysema with enlargement of the chest: the anteroposterior diameter is much increased.
Ward 6, Philadelphia Hospital. (Original.)

4. *The Chest of Disease of the Nervous System.*—In the boat-shaped chest found in syringomyelia the upper portion of the anterior part of the thorax is depressed forming a vertical groove that may be 5 cm. deep. Progressive muscular atrophy gives rise to a box-shaped thorax which stands out conspicuously above a collapsed abdomen.

5. *The Transverse (or Harrison's) Groove.*—This is a depression or groove which extends from the xiphoid cartilage sloping downward and slightly outward toward the axilla. It may be a developmental

FIG. 87



The flat or phthisical chest; short anteroposterior, long transverse diameter. (Gee.)

FIG. 88



FIG. 89



The phthisical chest. Full-blooded Indian, Philadelphia Hospital. (Original.)

fault, but in adults usually signifies respiratory obstruction or rachitis in early life. It marks the insertion of the diaphragm to the ribs.

III. 1. *The Flat Chest.*—The anteroposterior diameter is small and the transverse diameter increased. The costal angles are acute, the ribs and costal cartilages are flattened instead of having the normal convexity.

2. *The Alar Chest.*—The chest is elongated; the ribs are markedly oblique, causing a falling of the shoulder; hence the scapulae are prominent, often so markedly that the term alar or winged is given to this type. The neck is long, the larynx very prominent, and the arms elongated.

It is also known as the pithisical chest, but, as with the flat chest, this does not necessarily mean the patient has or will have tuberculosis. Individuals with such types of chest are more liable to contract the disease than those with normal chests, but may go through life to die of other diseases.

FIG. 90



Funnel breast (Trichterbrust). (Original.)

3. *The Funnel Chest.*—The lower end of the sternum forms a deep concavity. The condition is usually congenital, and is often seen in several members of a family (Warthin), but may be due to obstructed

respiration or may result from constant pressure, as when found in shoemakers.

UNILATERAL CHANGES IN SHAPE.—*Unilateral Enlargement.*—This usually is most prominent at the base. The length of the chest is increased; the ribs are elevated; the side is more rounded; and the costal angle is more obtuse. The interspaces are frequently effaced, or fuller than on the corresponding side. The nipple is displaced outward. The scapula of the affected side is also displaced outward; hence the distance from it to the spine is greater than on the opposite side. (See Fig. 91.)

The movements may be increased if the enlargement is due to an increase of normal contents of the lung as in compensatory emphysema or decreased if the cause is abnormal as air or fluid of the pleural sac.

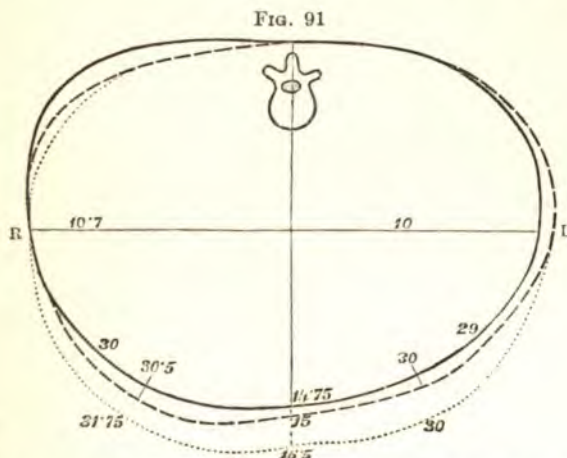


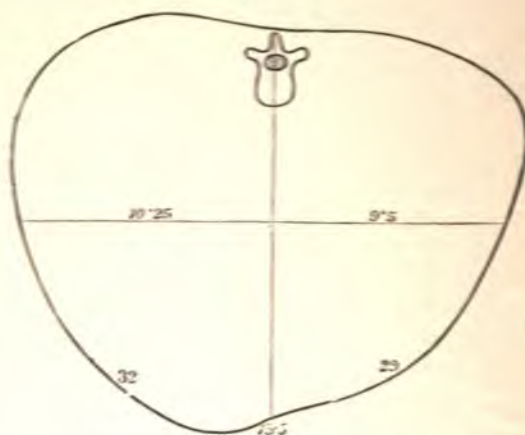
Fig. 91
Unilateral enlargement of chest (right side) artificially produced by injecting air into the right pleural cavity. Unbroken line: outline before injection. Broken line: outline after moderate distention. Dotted line: outline after extreme distention. Figures at bottom of vertical line indicate the anteroposterior diameter; along horizontal line, transverse semidiameter; remaining figures, right and left semicircumferences. (Gee.)

Unilateral Contraction or Diminution in Size.—The affected side looks flat before and behind, the anterior or the posterior portion, or both, being depressed. The semicircumference is lessened, as is the diameter through the nipple or any other fixed point. The costal angles are sharper. The ribs are closer together, and may almost overlap. The interspaces are lessened in width and may be drawn in. The movement of the side is lessened. Any diminution of contents will cause diminution of the affected side. Unilateral contraction is most frequently seen in cases of chronic pleurisy and of fibroid phthisis. It may occur from obstruction or compression of the bronchi of that side.

LOCAL CHANGES IN SIZE AND SHAPE.—Enlargements and diminutions seen in localized areas.

Local Enlargement.—That occurring in the region of the heart and great vessels, where it is particularly common, will be considered in the chapter on Diseases of the Heart and the Bloodvessels. A local enlargement in the lower anterior or lateral region of the chest may occur in cases of empyema, in which the pus tends to point externally, in cases of pulsating pleurisy, in enlargements of the liver or ascites.

FIG. 92



Unilateral retraction of chest, consequent upon cirrhosis of the left lung, in a girl, aged fourteen years. The figures indicate anteroposterior and transverse diameters and semi-circumferences of right and left half of the chest. (Gee.)

Local Contraction.—This may be seen either at the apex or at the base of the lung. At the apex it is seen above and below the clavicle. The interspaces are sunken and the ribs depressed. The term flattening is applied to this condition, which may be more readily seen when looked at from behind, downward. Flattening may also occur either in the lateral or in the posterior region at the base. The physical condition causing local contraction is the same as that causing unilateral or general contraction, or diminution in size of the structures within. This is notably seen in tuberculosis. Local pleurisy, with organization and contraction of the inflammatory exudate, may also cause diminution of the amount of air, or diminution of the contents, from compression of the adjacent lung structure. In local contractions *movement* of the part is generally diminished.

The Movements of the Chest.—The frequency, the rhythm, the type, and the character of the respirations, the degree of expansion, and the so-called diaphragm phenomenon are studied.

The Movements in Health.—A complete respiratory act consists of two events, inspiration and expiration; inspiration being active and expiration passive. The latter is a trifle longer than the former, as may be illustrated by the following proportion—Inspiration : Expira-

tion :: 5 : 6. A pause follows the act of expiration. In inspiration, as the lung expands with air, the chest increases in circumference and in length. The *degree of expansion* varies.

The Frequency and Character of the Movements in Health.—These vary in the two sexes. In a healthy male adult the respirations are from 16 to 24 in the minute. In the female they may be 20 to 22. In children the frequency of respiration is much greater. In those under one year it is 44 per minute; at five years it is 26. The respirations are increased in frequency in the standing position, during bodily exertion, with increased temperature of the air, and during digestion. They are lessened in the horizontal position. The hand placed on the epigastrium facilitates counting the respirations.

Normal Types of Breathing.—Two types of normal respiration are recognized. In the *abdominal* or *diaphragmatic type*, seen in male adults during quiet breathing, the movements of the chest are more marked in the lower half of the thorax and the upper abdomen. The *costal* or *upper thoracic type of breathing* is seen in women, in whom the upper half of the chest moves more actively. The *abdominal type* also occurs most frequently in children. It is the type of breathing in both sexes during sleep and is observed during deep respiration.

The Movement of the Chest in Disease.—In disease as in health the type, character, rate and rhythm of respiration, the degree of expansion and Litten's phenomenon are observed.

The Types of Respiration in Disease.—Any of the normal types of respiration may predominate abnormally or may be abnormally increased.

Abnormal costal breathing may be caused by edema or inflammation of the lower lobes of the lungs; double pleurisy with effusion; diseases of the bony thorax; painful affections of the soft parts of the lower thorax or upper abdomen; paralysis of the diaphragm; pressure on the diaphragm from ascites, tympanites, or from abdominal tumors or enlargements; diseases below the diaphragm, such as peritonitis; and by emphysema, and hysteria.

Abnormal abdominal breathing may occur in pleurisy, pleurodynia, fracture of a rib, calcification of the costal cartilages, scleroderma of the chest wall, ossifying myositis, and paralysis or spasm of the muscles of inspiration.

THE RESPIRATORY RATE IN DISEASE.—The rate of the movements is *increased* in nearly all forms of dyspnea. Slowing of the respiratory rate, *oligopnea* takes place in conditions of stupor and of coma. (See Dyspnea.)

ALTERATIONS IN THE CHARACTER AND RHYTHM OF RESPIRATION.—The normal ratio may be disturbed by an increase in the length of either inspiration or expiration, or an irregularity of the time and rhythm.

Inspiration Increased.—The duration of inspiration is increased when there is an obstruction in the trachea or larynx. It is accompanied by increased expansion of the upper chest, which is usually

associated with retraction of the soft parts of the thorax, especially at the base. The character of the breathing in such a case if the obstruction is in the larynx is a peculiar whistling, screechy, or shrill sound (stridulous respiration).

Expiration Prolonged.—In cases of asthma, chronic bronchitis, and emphysema the expiration is prolonged, and the muscles of expiration are seen to be brought into full action.

Stertorous breathing, snoring, is seen often in the unconsciousness of apoplectic, uremic, or diabetic coma and of narcotic poisoning; also in paralysis of the palate and in cases of enlarged faucial or pharyngeal tonsils. The prolonged expiratory sounds arise from the vibrations of the relaxed soft palate.

Irregular Breathing.—Irregularities in the respiratory force and in the intervals separating the respiratory cycle are seen in numerous conditions.

1. *Sighing.*—A slow and deep inspiration followed by a rapid and forced expiration, the pause intervening between the sigh and the normal rhythm being of normal occurrence, becomes abnormal if frequent and pronounced. It is seen in the hysterical and hypochondriacal and in conditions of low blood pressure as hemorrhage or shock as a physiological effort to more fully oxygenate the blood. It is seen quite commonly in typhoid fever and dilatation of the heart, more rarely in cerebral and meningeal disease.

2. *Jerky or Catchy Respiration.*—A spasmodic jerking of expiration or inspiration, or both, may occur in pleurodynia, fractured rib, fibrinous pleurisy, and sometimes in asthma, hydrophobia, and hysteria.

3. *Biot's (or Meningeal) Respiration.*—In this type of respiration common in cerebral meningitis there are regular, occasionally irregular, pauses in the breathing lasting for a few seconds to a half-minute.

4. *Cheyne-Stokes Respiration.*—The respirations are carried on by an alternation of pauses in respiration (period of apnea) and of periods of modified breathing (period of dyspnea). The respiratory pause may last for twenty-five to forty seconds, followed at first by shallow slow respiration which gradually gather rapidity and depth until the acme is attained, when they gradually decrease until the period of apnea is attained. Sometimes consciousness is lost during the pause, and often the pupils are contracted and inactive and the pulse-rate slowed. When the respirations begin the pupils dilate, consciousness returns, and the pulse is accelerated. This type of breathing usually indicates grave cardiac, renal, or cerebral disease, especially in arteriosclerotic individuals. Medicinal doses of morphine may intensify the phenomena or even bring it on.

ALTERATIONS IN THE DEGREE OF RESPIRATORY EXPANSION.—These may be (1) bilateral, (2) unilateral, or (3) local.

1. *Bilateral Changes in Movements.*—*Increased expansion* occurs normally after exercise or mental excitement, and is often seen in hysteria and in some forms of dyspnea.

Diminished expansion may be a part of a condition of general muscular weakness, or it may be due to paralysis or spasm of the respiratory muscles, to obstruction of the upper air passages or to emphysema; it may be caused by an effort to limit the pain of pleurisy, pleurodynia, or fractured rib.

Inspiratory retraction of the lower ribs is found at times in emphysema, ossification of the costal cartilages, and visceroptosis.

2. *Unilateral Changes in Movements.*—*Increased unilateral movement* is seen when the lung of one side is acting vigorously to compensate for the other lung which is disabled by disease. The whole side moves more rapidly and vigorously. The increased movement is associated with enlargement of the affected side and hyperresonance on percussion.

Diminished unilateral movement occurs because movement is inhibited on account of the pain of pleurisy or intercostal muscular rheumatism or fracture of the ribs; because less air enters the lung as in occlusion of a bronchus by a foreign body or by the pressure of an aneurism or mediastinal tumor; because there is less air space on account of consolidation, as in pneumonia or pleurisy; or because of compression of the lung from air or fluid in the pleura or by adhesion of the pleura.

3. *Local Diminution of the Respiratory Movement or Deficient Expansion.*—It occurs under the same conditions, tuberculosis (infiltration or cavity), local pleurisy, etc., as produce flattened and local contractions, and for the same reason.

FIG. 93



Litten's diaphragm phenomenon.

LITTEN'S DIAPHRAGM PHENOMENON.—In persons whose chest walls are not too thick the movements of the diaphragm are indicated on the surface of the chest by the rise and fall of a shadowy line. The patient must lie on his back with his face turned away from the light and with the head slightly elevated. The light should fall from the head or foot of the bed. The observer stands a distance of three or four feet away, with his back to the light, scanning the chest at an angle

of about 45 degrees. In the act of inspiration a horizontal shadow or undulation is seen to start on either side about the sixth interspace and to pass during inspiration downward over a distance of two or more interspaces, even to the margin of the ribs. In expiration the shadow begins below and moves upward to the starting-point. By this phenomenon the volume or vital capacity of the lungs can be estimated. In normal individuals the shadow should move more than 6 cm.

The Change in Litten's Phenomenon.—Absence of the phenomenon is noted: when the pleural cavity contains fluid or air, and when it is obliterated by adhesions; in pneumonia of the lower lobe; in emphysema of the lungs; and in intrathoracic tumors low down in the chest. Tumors or fluid accumulations below the diaphragm do not lessen the phenomenon. With lessening of the extent of movement the respiratory capacity is diminished and tuberculosis may be suspected. Limitation of the excursion of the diaphragm is one of the earliest signs of tuberculosis. This limited excursion can be detected in proper subjects by Litten's method, although it must be remembered that general debility and emphysema lessen the excursion on both sides. In splenic and hepatic enlargements the normal shadow persists, but in a large collection of ascitic fluid it may be detected with difficulty or may be absent.

Thoracometry, Cyrtometry, Spirometry, Pneumatometry, and Stethography.—The result obtained by inspection can be confirmed by certain instruments of precision.

Thoracometry or Mensuration.—By this means the size and the degree of expansion of the chest are ascertained and the circumference and diameter of the chest are determined.

The diameter of the thorax is measured by means of caliper compasses. The average anteroposterior diameter, on a level with the nipple anteriorly and without the insertion of the seventh rib posteriorly, is 19 cm. in men and 17 cm. in women. The transverse diameter at the highest point in the axilla is 25 cm. in males. The average circumference of the chest in men is 85 cm.; in women 74 cm.

The *respiratory capacity* is estimated by measuring the circumference of the chest at the level of the nipple. It is secured by taking the measurement at the end of complete expiration and again at the end of complete inspiration. In health the difference between the two measurements should be from 5 to 10 cm. (2 to 4 inches). The expansion is less in women. Deficiency of chest expansion not only indicates the presence of a local morbid process (notably incipient tuberculosis), but it also indicates lack of strength and of muscular development, physiological rather than physical deficiencies, and is an unerring guide to the need of respiratory gymnastics.

Cyrtometry.—This is the determination of the shape of the chest. By this means differences in the shape and in the movements of the

two sides are made manifest. An easy and simple method is to take two flexible lead strips. One strip is moulded to fit the anterior surface of one side of the chest, the other strip the posterior surface, the two pieces meeting at the midaxillary line. The excess of the strips which may reach past the midline is bent sharply back so that each piece extends only from the midline to the midaxilla. The two pieces are then removed and the outlines traced on a piece of paper. A similar procedure is carried out on the opposite side.

Spirometry.—The object of spirometry is to ascertain the respiratory capacity of the lungs by means of a specific instrument, the spirometer. The average lung capacity (the amount of air exhaled after a full inspiration) is 325 cubic inches for each inch of height; 23.19 c.c. for each centimeter (Otis).

Pneumatometry.—This consists of measuring by a special instrument the force of expiration and inspiration. The method is of very little diagnostic value.

FIG. 94



Noting expansion and vocal fremitus.

Stethography.—This is the graphic method of recording movements of the chest during respiration.

Palpation of the Chest.—Palpation in diseases of the lung and pleura is employed (1) to confirm the results obtained by inspection, mensuration, and cyrtometry as to the size, form, and movements of the chest; (2) to elicit tenderness; (3) to determine the resistance of the chest wall and of tumors; (4) to distinguish the vibration produced by the voice (vocal fremitus), by bronchial rhonchi (rhonchal fremitus), and by pleural friction (friction fremitus); and (5) to detect the fluctuation caused by fluid and the succussion due to the presence of both fluid and air.

Method.—The surface should be bared, although the fremitus can be detected through a thin layer of linen or gauze. To estimate the degree of expansion and to detect the fremitus in front, it is often well

are thus transmitted transversely through the lung tissue; this is particularly marked at the right apex where the fremitus is greater

FIG. 96



Testing the vocal fremitus with the edge of the hand.

FIG. 97



Vocal fremitus.

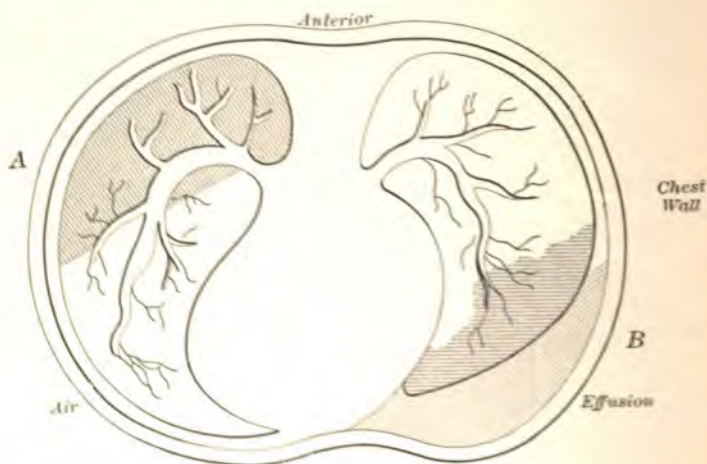
than in any other portion of the chest. It is stronger in persons with deep, low-pitched voices, the vibrations not being so rapid, and is

more distinct therefore in males than in females. It is felt more distinctly in persons with thin chest wall and is greater between the scapulæ. *Fremitus* is *weaker* in people with thick chest walls and large mammary glands which interfere with the transmission of *fremitus*. The *fremitus* is not distinct in children because the vibrations are too rapid.

VOCAL FREMITUS IN DISEASE.—The vocal *fremitus* may be increased, diminished, or absent. To obtain *fremitus* it is necessary to have a sufficiently resonant voice, patulous bronchi, and relatively free transmission from the lungs to the chest wall.

Increased Vocal Fremitus.—Vibrations are transmitted to the hand with greater force by a solidified lung than by one containing air. *Fremitus* is increased consequently in pneumonia, tuberculosis, hemorrhagic infarct, and tumor. The *fremitus*, however, may be absent

FIG. 98



Transverse section of thorax: A, solidification—pneumonia, vocal *fremitus* increased; B, pleural effusion, vocal *fremitus* absent. (Original.)

in the rare cases of pneumonia in which the large tubes are occluded by exudate. *Fremitus* is increased when the lung is compressed above a pleuritic exudate. It is also increased over a cavity, which acts as a resonator.

Diminished Vocal Fremitus.—Anything intervening between the lung and the surface of the chest and interfering with the conduction of the vibrations diminishes the *fremitus*. The *fremitus* therefore is diminished over a thickened pleura and over a thin layer of pleural effusion. The *fremitus* is also lessened when on account of diminution in the caliber of the bronchi the columns of air in them are smaller, as in bronchitis, in emphysema, and in asthma. The *fremitus* is less

over cavities filled with fluid and over a lung whose bronchus is partially occluded.

Absent Vocal Fremitus.—The vocal fremitus is absent when the columns of air are entirely obstructed by occlusion of the bronchus, as by the external pressure of a tumor, aneurism, or enlarged glands, and when the pleura contains air or fluid, causing interference with the vibrations (Fig. 98), as in pneumothorax, hydrothorax, pyothorax, and hemothorax. When the pleura is greatly thickened, the fremitus is absent.

Bronchial Fremitus.—The vibrations produced by the passage of air through mucus or fluid in the bronchial tubes may be transmitted to the hand when it is laid on the surface of the chest, and constitute the *rhonchal* or *bronchial fremitus*. They are felt during inspiration in bronchitis and asthma. In phthisis, air passing through fluid in a cavity may produce vibrations over a localized area. In children with bronchitis, rhonchi are distinct, and are often the source of much alarm to parents.

Friction Fremitus.—A rubbing vibration which is transmitted to the hand and which occurs synchronously with respiration or with the heart-beat is known as a friction fremitus. Bronchial fremitus is influenced by cough and by breathing, a friction fremitus is not.

Fluctuation.—This may be detected by palpation in some cases of effusion, particularly if the intercostal spaces are swollen and tense, and in the case of an empyema about to point.

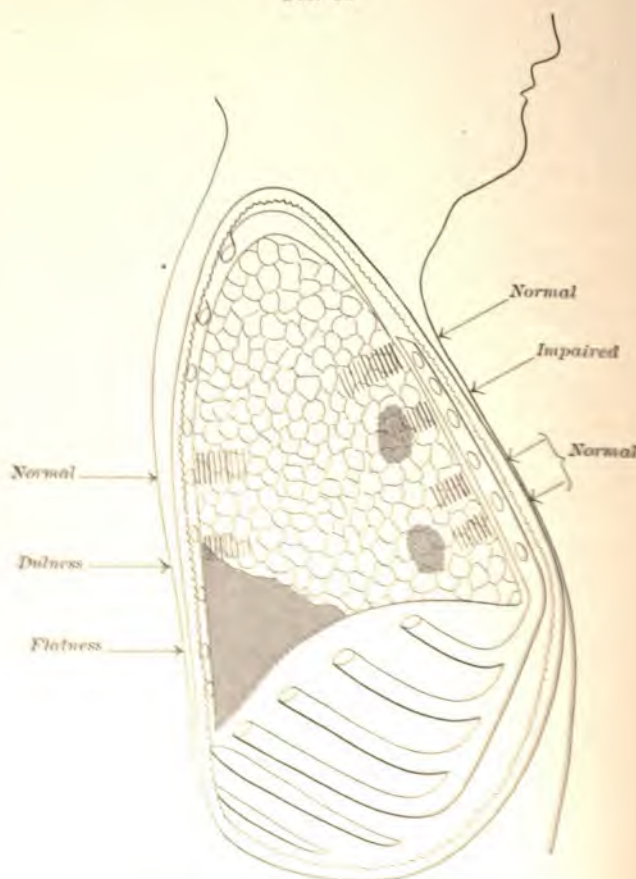
Splashing or Succussion.—When both air and fluid are present in the pleural cavity, or in a large pulmonary cavity situated close to the thoracic wall—on shaking the chest the motion of the liquid may be felt by the palpating hand.

Percussion in Diseases of the Lungs and Pleura.—Percussion is employed in disease (1) to elicit abnormal sounds, and (2) to define boundaries.

Modifications of the Sounds in Health.—The degree of clearness or resonance differs in various parts of the thorax. It is purer in the upper axillary region, at the angle of the scapula, and on the anterior surface of the chest, in the second interspace. It is slightly higher in pitch at the right apex than at the left. It has a tympanitic quality between and over the sternal ends of the clavicle and at the left base. It is modified by the condition of the chest wall. Thick chest walls, accumulations of fat, the mammary gland, and the scapulæ impair the resonance and necessitate deep percussion to bring out the true sounds. In persons with thin chest walls the resonance is clear and more pronounced. The percussion note is also modified by the elasticity of the chest walls. In the aged, because of rigid chest walls, it is less clear. In children in whom the chest walls are elastic the resonance is much fuller or clearer. The sounds therefore vary, within certain limits, in different individuals with perfectly healthy, normal chests. Moreover, a sound normal in one part of the chest may in another part indicate disease.

Percussion sounds have consequently no absolute value; their significance depends upon the individual and upon the part of the chest examined. The student should learn from the outset to compare the sounds developed by percussion of symmetrical portions of the chest and thus determine the normal for the individual.

FIG. 99



Changes in percussion note. (Le Fevre.)

Spinal Percussion. Healthy vertebrae give rise to characteristic percussion sounds depending on the character of the adjacent bony structures. We find normally dulness over the 1-IV thoracic vertebrae; resonance over the V-XII thoracic vertebrae; impaired resonance over the lumbar vertebrae; flat tympany over the sacral vertebrae. Thoracic dulness is shortened in emphysema and lengthened in aneurism or mediastinal growths. Pulmonary solidifications

the thoracic resonance. An effusion elevates the lower limit of thoracic resonance in sitting or standing postures.

Abnormal Percussion Sounds.—The percussion sound elicited in disease may be (a) *higher pitched* than normal, (b) *clearer* than normal, or (c) of a *different character* from the normal sound. To the first group belong *diminished* or *impaired resonance*, *dulness*, and *flatness*; to the second, *hyperresonance* and *tympany*; and to the third, *amphoric resonance*, the *cracked-pot sound*, *Williams' tracheal tone*, the *bell or anvil sound*, *Wintrich's phenomenon*, *interrupted Wintrich's phenomenon*, *Friedreich's phenomenon*, *Gerhardt's phenomenon*, and *Biermer's phenomenon*.

It may be said in general that when a percussion sound is produced which varies from the normal resonant tone, it indicates an abnormal physical condition. In percussing the chest, however, exactly corresponding portions of the two sides must be compared. Both light and deep percussion should be employed over every part of the lung.

Change in tone may be (1) *bilateral*; (2) *unilateral*; or (3) *local*.

DIMINISHED OR IMPAIRED RESONANCE.—The normal tone or resonance is impaired, or muffled in cases of beginning solidification of the lung, in slight thickening of the pleura, and in small pleural effusions which overlap the lung as a thin layer. It is the first change toward dulness. It is particularly noted in the early stages of phthisis, when the lung usually is the seat of small areas of tuberculous infiltration at the apex.

DULNESS.—The dull note signifies a relatively small amount of air in proportion to solid structure, the extent and the degree of dulness depending upon the proportion of solid to air-containing material. Moderate dulness is seen in moderate tuberculous infiltration of the lung, and in small patches of catarrhal pneumonia, in pulmonary congestion and edema, in phthisis, in condensation of the lung from pressure, in carcinomatous infiltration, in atelectasis, in the presence within the pleural sac of serum, pus, or lymph not sufficient to cause flatness and in the intrascapular region over enlarged bronchial glands, a mediastinal tumor or aneurism.

FLATNESS.—Absolute or complete dulness, flatness, occurs when air is completely absent, as in the stage of hepatization of acute pneumonia, in hemorrhagic infarction, in the solidifications of phthisis, in interstitial pneumonia, in carcinoma of the lung, in pulmonary abscess, in filled phthysical cavities or circumscribed gangrene, in condensation from pressure—by pleurisy with large effusion, in empyema, in a large hydrothorax, in great thickening of the pleura, and in solid tumors.

HYPERRESONANCE.—When the resonance is increased, the sound is abnormally clear. If it is fuller and clearer than in health, without the characteristics of the tympanitic note, it is known as hyperresonance or *exaggerated resonance*. The physical condition that causes exaggerated or hyperresonance is increase in the amount of air. This increased amount of air may be *general*, *unilateral* or *local*. When general

(*bilateral*) it gives the characteristic sound heard in emphysema. A like increase in resonance may be present in acute miliary tuberculosis.

Unilateral increase in resonance occurs when there is an increased amount of air in one lung on account of compensatory enlargement (vicarious or compensatory emphysema), or on account of an increase of air in the pleura. *Local* increase of resonance occurs when a local area of the lung is acting in a compensatory manner. This is seen in cases of phthisis in which the alveoli or lobules surrounding small areas of consolidation are greatly distended. The exaggerated note may aid in the recognition of a deep solidified area. *Skodiac resonance*, a clear, high-pitched percussion note, is obtained over a portion of the lung above the line of pleural effusion, and above the line of solidification in pneumonia, being due to relaxation of the lung tissue.

TYMPANY IN DISEASE.—Tympany signifies that air is confined in a large space (cavity) or that there is an excess of air in many sacs (emphysema). A tympanitic sound from the chest occurs: (1) *bilaterally*, in cases of marked emphysema, and (2) *unilaterally* in cases of pneumothorax and marked compensatory emphysema. (3) Tympany may also occur *locally*: (a) in some cases of local compensatory emphysema; (b) in the early stages of pneumonia, or in the later stages of complete solidification; (c) in cases of pleural effusion, owing to alteration in the tension of the lung, a tympanitic note is present above the layer of fluid; (d) in phthisical cavities at the base or the apex, and in bronchial dilatation; if the cavity communicates with the air, has moderately thin, elastic walls, and is at the same time empty, a tympanitic note is produced.

SPECIAL SOUNDS.—*Amphoric resonance* and *cracked-pot* sounds are discussed under Percussion (Chapter XIX, page 228).

Williams' Tracheal Tone.—This is a tympanitic sound elicited over the apex of a solidified, shrunken, or thickened lung when it is percussed in front. It is due to the vibrations produced by the percussion blow in the trachea and primary bronchi, which vibrations are conducted by the solid lung tissue.

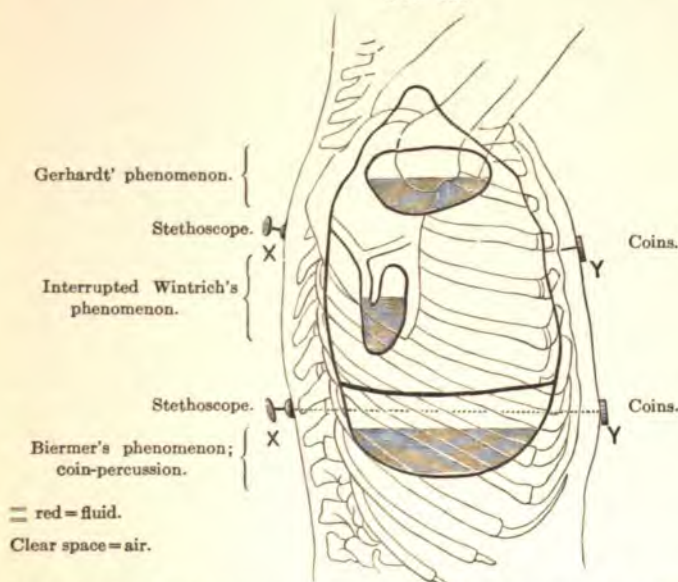
Wintrich's Change of Sound.—The sound elicited by percussion over a cavity communicating with a large bronchus changes when the patient alternately opens and closes his mouth, becoming louder, more distinctly tympanitic, and higher in pitch when the mouth is open.

Interrupted Wintrich's Change of Sound.—Wintrich's phenomenon may be distinct in some positions of the body, but indistinct or absent in others. This is a positive sign of a cavity containing fluid which occludes the communicating bronchus in one position but leaves it open in the other.

Friedreich's Respiratory Change of Sound.—The note over a cavity is higher in pitch at the end of inspiration than after expiration. This is due to increased tension of the chest wall, of the lung tissue, and of the walls of the cavity, and to the widening of the glottis during inspiration.

Gerhardt's Change of Sound.—The tympanitic sound elicited over a cavity containing fluid may change its pitch with change of the position of the patient, especially if the cavity be longer in one diameter than

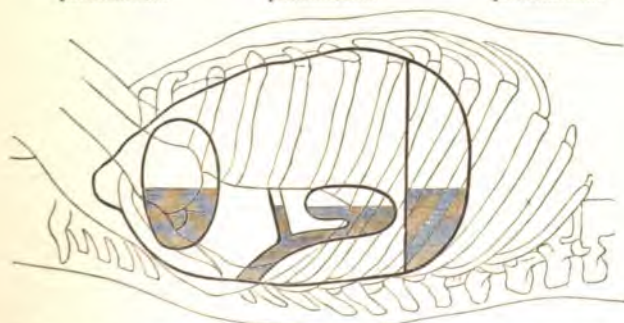
FIG 100



Illustrating Gerhardt's and Biermer's phenomena, interrupted Wintrich's phenomenon, and coin-percussion.

FIG. 101

Gerhardt's phenomenon. Interrupted Wintrich's phenomenon. Biermer's phenomenon.



Illustrating Gerhardt's and Biermer's phenomena and interrupted Wintrich's phenomenon.

in the others. This is due to the change in the relative positions of the air and the fluid contained in the cavity.

Biermer's Change of Sound.—The percussion note over a pneumo-hydrothorax changes in pitch with alteration of the patient's position,

on account of the change in the relative position of the air, and the fluid.

Bell Tympany.—This is elicited in pneumothorax by *coin-percussion*. One person listens at the back of the chest while a second person percusses the front of the chest with two large coins, using one as a pleximeter and the edge of the other as a plessor. Air in the pleural cavity will conduct the sound so that it is heard on the opposite side of the chest as a soft, musical, metallic, echoing sound, like the chiming of a distant church bell or the ring of a hammer on an anvil far off.

The Lung-reflex.—It sometimes occurs that the more a dull area is percussed, the more resonant it becomes. Cabot believes this is part of the lung-reflex described by Abrams, who found that if an irritant such as cold or mustard be applied to any part of the skin covering the chest, the underlying lung expands in response to the irritation, producing a temporary localized emphysema.

Auscultation in Diseases of the Lungs and Pleura.—In auscultating the lungs we listen for (1) the sounds produced by respiration as heard over the larynx, trachea, and chest (the *breath-sounds*); (2) the new or *adventitious sounds* present only in disease; and (3) the resonance of the spoken voice as heard over the chest (*vocal resonance*).

In listening to the respiratory sounds their character must be noted in inspiration and in expiration and the relative lengths of the two must be observed during normal and exaggerated breathing and after coughing. In health the movements of inspiration and of expiration are almost equal; but the sound of inspiration is heard during the entire inspiratory movement, while that of expiration occupies only the first third or so of the expiratory act. The sound produced during expiration may even be less than half the length of that produced during inspiration. The following proportion represents relative lengths—Inspiration : Expiration : : 3 : 1.

The Breath-sounds in Health.—These include the two normal sounds and their modifications in health.

BRONCHIAL BREATHING OR NORMAL LARYNGEAL OR TRACHEAL RESPIRATION.—If the stethoscope be placed over the trachea at the top of the sternum, a sound will be heard characterized as follows: (a) it attends both inspiration and expiration with a definite pause between, (b) the inspiration and the expiration are nearly equal in length, (c) it is of a tubular, blowing character and relatively somewhat high-pitched, and (d) there is an absence of normal vesicular elements. The expiration is perhaps a little stronger and longer than the inspiration. The sound that is heard in this situation is known as *bronchial breathing*, and is one of the normal sounds of the chest. It may be heard behind, over the sixth and seventh cervical vertebræ.

The sound may be imitated by breathing deeply with the back of the tongue and the soft palate in the position to pronounce the guttural "ch" or "h."

Cause of Bronchial Breathing.—The sound is caused by the passage of air through the nares into the wider pharynx when the mouth is closed, and through the trachea and large bronchial tubes.

VESICULAR BREATHING, OR THE NORMAL RESPIRATORY MURMUR.—If the ear be applied over the anterior portion of the chest, or below the angle of the scapula behind, a sound is heard both on inspiration and expiration, differing from bronchial breathing, however, in that inspiration and expiration are changed in length. The sound of inspiration is twice or three times as long as the sound of expiration.

The sound of inspiration is soft, breezy, or sighing in character, increasing in intensity to the end of full inspiration. It is immediately followed by expiration, which diminishes in intensity as the air is expelled, and terminates when one-half or two-thirds of the expiratory act is completed. The sounds can be imitated by breathing with the lips in the position required to pronounce "f" or "v."

The vesicular murmur is produced partly in the finest bronchial tubes and air cells by their expansion and contraction and partly in the upper air-passages, the sound produced in the latter being modified on account of the intervention of the air-vesicles between the ear and the larger bronchi.

BRONCHOVESICULAR BREATHING.—In this type of respiration are combined in varying proportion the character of the bronchial and normal vesicular breathing. It is supposed to be midway between these two types of breathing and hence is known as transitional or mixed breathing, though actually one type may be much more pronounced than the other. It is normally heard anteriorly over the lower part of the manubrium and on each side of it for a slight distance, as well as almost invariably at the right apex. Posteriorly it is heard in the interscapular region at the level of the second, third, and fourth vertebræ. It occurs over the primary bronchi which are covered by a thin layer of lung tissue, thus combining both the bronchial and vesicular elements of respiration.

MODIFICATIONS OF THE BREATH-SOUNDS IN HEALTH.—The normal breathing-sounds may be exaggerated or may be diminished in intensity.

Exaggerated Breath-sounds.—The breath-sounds are increased in loudness and sharpness by strong, rapid breathing. The sounds are increased in children, in whom are combined greater elasticity of the chest wall and greater friction throughout the smaller bronchi, which are relatively larger. So distinct and characteristic is the sound in children that the term *puerile* respiration is applied to it. The sounds of inspiration and expiration are both intensified or sharper than in healthy adults, that of expiration being relatively prolonged.

Feeble Breath-sounds.—The sounds are modified by the condition of the chest walls. If they are thick, or there is an abundance of fat, the sounds are fainter or lessened in intensity. In wasting and exhausting diseases, feeble respiratory power causes feeble breath-sounds. The condition of the upper air-passages, even if not pathological,

modifies the sound. If the glottis is small, or the relationship between the nose and pharynx is disturbed, the sounds will be modified—they are usually weakened.

The Breath-sounds in Disease.—It is well for the student to bear in mind that normal sounds heard in positions which they do not normally occupy always indicate disease as well as sounds heard in the chest which are departures from the normal sound.

The normal vesicular murmur may be altered (1) in intensity, (2) in rhythm, and (3) in character.

Increased Vesicular Breathing.—*Bilaterally* the vesicular breathing or respiratory murmur is increased when there is increase in the force of breathing, when normal respiration is increased and the patient takes full, deep breaths; also in some forms of dyspnea, as at the acme of the Cheyne-Stokes breathing, or in the dyspnea of diabetic coma, and in certain forms of bronchitis, particularly when the small tubes are narrowed by inflammatory swelling.

Unilateral exaggeration or increase of vesicular breathing is heard when one lung is acting vigorously or in a compensatory manner.

Local exaggeration of vesicular breathing is heard over the apex when pneumonia or pleurisy is affecting the base of the lung, and *vice versa*.

Diminished or Absent Vesicular Breathing.—It occurs *bilaterally* in a number of conditions:

1. The normal vesicular murmur is lessened in all cases in which expansion is interfered with, as in the feeble; particularly at the bases posteriorly, if the muscles of respiration are paralyzed and if expansion is interfered with on account of disease of the diaphragm or by pressure upward by ascites.

2. Vesicular breathing is diminished by anything that lessens the amount of air supplied to the chest, such as occlusions or obstruction of the nares, of the pharynx, or of the larynx.

3. Chest walls thickened from disease, as in edema, weaken the respiratory sound.

4. The vesicular breathing is weakened throughout the entire extent in congestion and edema of the lungs. These conditions cause a feeble respiratory murmur except at the anterior margins of the lungs.

Unilateral diminution of breath-sounds occurs: (1) When there is narrowing of the bronchus, from pressure of an aneurism or a mediastinal tumor.

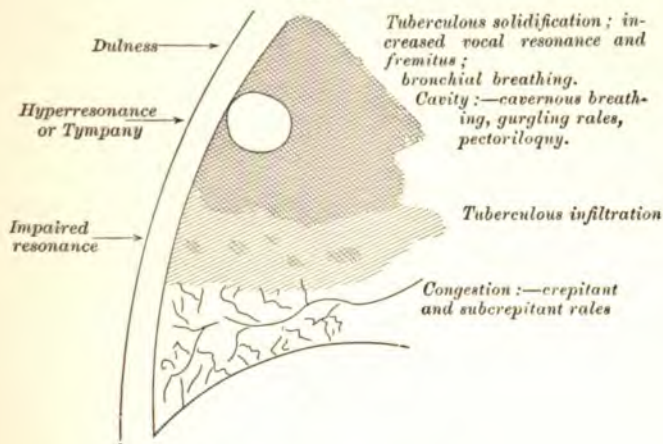
2. When there is pleural effusion, which (a) lessens the amount of air-pressure by compressing the lung and (b) interferes as a different conducting medium. (See Fig. 98.) If the pain of pleurisy, pleurodynia, or neuralgia limits expansion of one side, the breath-sounds of the affected side will be lessened. Not only in pleural effusions but also in thickened pleura there is weakness or faintness of the respiratory murmur. It should not be forgotten that effusions and thickenings of the pleura rarely take place bilaterally.

Local diminution of breath-sounds occurs in the early stages of phthisis or in the earliest stages of pneumonia.

ALTERATION IN RHYTHM OF THE BREATH-SOUNDS.—The only changes in rhythm, having a distinct clinical value, are prolonged expiration and jerking inspiration.

Prolonged Expiration.—By this is meant prolongation of the expiratory murmur, so that it equals or is even longer than the inspiratory murmur. This is due to the difficulty of the air in getting out of the chest in consequence of loss of elasticity of the cells, or of an obstruction in the bronchi. Hence, prolongation of expiration all over the chest is seen in emphysema, particularly with bronchitis and asthma. A local area of slightly prolonged expiration or bronchovesicular breathing is one of the first physical signs of tuberculosis, and is particularly suggestive if occurring at the left apex.

FIG. 102



Various stages of phthisis.

Jerking or Interrupted Inspiration (Cog-wheel Breathing).—The inspiratory sounds may be created in puffs or jerks, so that during the act of inspiration, as the chest expands, a number of successive vesicular sounds are heard until the act is completed. The condition occurs in the earliest stages of tuberculosis, when the various bronchioles are more or less occluded by outgrowths of tubercles or by tenacious mucus. The air, entering different lobules at different periods of time, thereby gives rise to this peculiar broken sound. This must not be confounded with the same character of breathing heard near the heart, due to pressure exerted upon portions of the lung by the heart or by structures in intimate relation with it, on account of which air enters various areas in puffs. Jerking inspiration sometimes occurs in health,

and is simulated by the jerky act of inspiration in nervous patients. It is of no significance unless attended by other physical signs.

In cases of adhesion at the apex, particularly of the left lung, the same puffing or jerking inspiration is often heard. When pathological jerking breathing is present, the expiration is prolonged; and if the case is under observation a sufficiently long time, bronchial breathing will usually replace the jerky respiratory murmur through progressive solidifications. Small moist rales, excited by coughing or a full breath, usually attend jerking breathing when it is pathological.

ALTERATION IN CHARACTER.—The normal vesicular murmur may be altered in character, becoming harsher, and rougher.

BRONCHIAL BREATHING.—The normal situation of bronchial breathing in health has been stated. If the same kind of breathing is heard in any other portion of the lung, it is pathological. It is generally indicative of solidification, the lung tissue being replaced by solid conducting material, which conducts the bronchial sound to the ear. It is the typical form of breathing heard in solidification of the lung due to pneumonia or tuberculosis, in hemorrhagic infarcts, and in lung syphilis, if a large bronchus is not occluded. In a central pneumonia where the solidification is deep-seated and surrounded by lung tissue bronchial breathing may not be heard, or it may be postponed until the third or fourth day of the disease.

In certain cases of pleurisy with effusion bronchial breathing exists. The accumulation of fluid not being sufficiently great to compress the lung completely, the bronchial tubes remain patent while the vesicular structure is compressed. Under these circumstances low-pitched bronchial breathing is heard, more pronounced over the upper layer of the effusion. It is always heard close to the spine posteriorly, when the lung is compressed. Sometimes it is heard above the limit of the effusion, in all probability because of relaxed tension of the lung.

Bronchial breathing also occurs in tumor of the lungs, as in pulmonary carcinoma.

Modifications of Bronchial Breathing.—While its special characteristics must be borne in mind, it must not be forgotten that bronchial breathing is not represented accurately in every instance by the sounds heard over the trachea. Its character may be modified and yet approach that type of breathing. The modification occurs in one or both of the two portions that go to make up the sound:

1. The blowing element may not be so distinct in inspiration as in expiration.

2. The characteristic blowing sound, in some cases, may continue so long during expiration as to equal in length the inspiratory sound.

3. Bronchial breathing may vary in pitch. (a) At times the sound is high in pitch in both inspiration and expiration, but with a pure harsh, blowing quality attending each. (b) It may be soft and low in pitch during both acts.

4. The loudness of the sound may also vary. This depends largely upon physical peculiarities of the individual, and is determined by the condition of the chest walls and the force of the breathing. When pleurisy with effusion coexists with pneumonia, the bronchial breathing, which should be audible, is feeble and distant.

Varieties of Bronchial Breathing.—The quality of bronchial breathing is altered in the presence of a cavity, several varieties being observed.

Cavernous Breathing.—If a case of tuberculous solidification is watched, it will be found after a time that the bronchial breathing becomes lower in pitch. It is heard in inspiration and expiration, but a more hollow quality attends the sound. From the hollowness of the tone the word cavernous has been applied to the breath-sound; it is due to the formation of a cavity in the diseased lung or to a dilated bronchus.

Amphoric Breathing.—Cavernous breathing may have a metallic quality and is then called amphoric. It resembles the sound produced by blowing across the open mouth of a jar. A large cavity with smooth walls that communicates with the air through a small opening is the cause of the development of such a sound. It is heard also in pneumothorax when such communication exists. The metallic tone is analogous to the metallic percussion-sound. It occurs under the same physical circumstances.

Metamorphosing Breathing.—Seitz has called attention to a form of breathing heard in connection with cavities, called the metamorphosing breath-sound. In this type inspiration begins harshly bronchial and then becomes faintly bronchial, the bronchial-sound being heard also in expiration. It is said to be a sure sign of cavity.

BRONCHOVESICULAR BREATHING.—The physical conditions producing bronchovesicular breathing are those that produce bronchial breathing only of a more moderate degree and a lesser amount. Thus it is heard over the small patches of solidifications of phthisis and bronchopneumonia, in the early and late stages of lobar pneumonia; in bronchitis associated with marked swelling of the mucous membrane; and in moderate condensation of the lung from pressure. Breathing of this type occupies a broad field and includes all pathological conditions in which the breath-sounds possess a vesicular element, yet which suggest the bronchial type of breathing.

New or Adventitious Sounds.—The foregoing sounds are modifications of the normal sounds heard during the act of breathing. New or adventitious sounds are also heard in the lungs or in the pleural cavity. They include rales, friction sounds, the succussion sounds and metallic tinkling.

RALES.—These are adventitious sounds, occurring only during respiration, appreciated by auscultation. They are primarily classified according to the chief characteristic sound appreciated by the auscultatory ear, viz., *sibilant*, *sonorous*, *crepitant*, *subcrepitant*, or *crackling*, and *bubbling*.

Sibilant rales are created in the small tubes and are high-pitched, whistling, or musical sounds.

Sonorous rales, created in the large bronchial tubes, are coarse, low-pitched musical sounds. Both these two forms of rales are heard in the early stages of bronchitis, in asthma and in the chronic bronchitis of emphysema. They are caused by the passage of air through tubes partially occluded by tough, thick mucus or by swelling of the mucous membrane. To these two factors may be added a third, a partial stenosis of the tube by muscular spasm.

Crepitant rales are fine distinctly localized rales, resembling the sound produced by rubbing a lock of hair between the fingers, and are heard only during inspiration. They are found in these pathological conditions in which there is present fluid in the alveoli, notably the early stages of pneumonia and edema of the lungs, occasionally in hemorrhagic infarction and acute pneumonic phthisis. They are created in the alveoli by inspiratory separation of alveolar walls, collapsed or held together by a sticky exudate.

Crackling or subcrepitant rales are of a somewhat larger size and lower pitched than crepitant rales. They are heard both during inspiration and expiration. The element of moisture is pronounced and is found in those conditions in which there is an excess of fluid in the terminal air passages, *e. g.*, diffusely in the second stage of bronchitis, edema, and hemorrhage of the upper air passages when blood is aspirated into the finer tubes; locally in phthisis, the early and late stages of pneumonia, and around any solidification with an attending congestion, edema or catarrh. They are created in the terminal air passages, by an abundance of secretion which is thrown into vibration by the current of air during the act of breathing.

Bubbling, gurgling, or mucus rales are coarse, low-pitched rales occurring in the larger bronchial tubes, or in cavities, from the same causes that produce subcrepitant rales. They are heard both in inspiration and expiration, and are very characteristic after a full breath or cough.

They are further qualified by their apparent size as medium, large, or coarse, according as they occur in small or large bronchi or cavities. While sometimes present in bronchitis and bronchiectasis, they are heard in their most marked form in the stage of softening of phthisis.

Rhonchi is a term frequently applied to musical sounds, produced in the large tubes, exemplified by sonorous and sibilant rales.

Wheezing is a term applied to palpable rhonchi which can be heard by the unaided ear, frequently some distance from the patient. Sonorous and sibilant rales are also known by the term dry rales, a misnomer, if understood to mean an absence of fluid or of increased swelling of the mucous membrane, as it is almost impossible to "conceive of a rale that does not to some extent at least, depend theoretically on moisture or increased turgescence for its causation." At the same time the term moist, referring to bubbling, gurgling, or subcrepitant rales, should be avoided, as all rales originate in or are caused by moisture.

Rales occasionally assume, a *consonating*, *metallic*, or *cavernous* quality if produced in pulmonary cavities with smooth, tense walls. In describing rales it is always well to note not only their auditory characteristics, but also their position, origin, and time of occurrence.

DIAGNOSIS OF RALES.—Rales are to be distinguished from other adventitious sounds. Although in some instances it is impossible to distinguish them from friction-sounds, as when rales are heard over the bases of the lungs, they nevertheless have certain marked characteristics. Rales are recognized (1) by the *qualities* previously mentioned; (2) by their *location*; if the adventitious sounds are general, they are due to rales; (3) rales are *modified* by *cough* and *breathing*. They may be intensified by either act, or after the completion of the act may disappear entirely. On quiet breathing, in the early stages of tuberculosis, for instance, they may not be heard at all. Before excluding them it is absolutely necessary to have the patient cough and then take a full breath; (4) they *vary* in *position*. This may occur from hour to hour. If the chest is examined in the morning, they may be more pronounced at the base for instance. At another time in the twenty-four hours they may be more distinct at the apex. They are more likely to be present at the base if the patient is kept in the recumbent posture; (5) they *vary* in *character*. At one time small, moist rales may be heard, and in a short time they may be replaced by larger rales. Sibilant and sonorous rales are regularly followed by mucus rales in the course of bronchitis. In a case of bronchial asthma all sorts of rales may be heard in a few hours. Rales are distant to the listening ear—they seem to be farther away than do the friction-sounds.

Rales in the bronchi must not be confounded with the crepitant or fine crackling sound which is heard at the base of the lung in patients who have been ill with exhausting fevers and who have not taken full breaths for some time. The latter disappear after the patient has inspired deeply half a dozen times.

Rales throughout the lung in themselves are not diagnostic of any affection save bronchitis, but their occurrence all over the chest is significant in the absence of other physical signs. In the absence of bronchitis, rales at the bases of both lungs are usually due to congestion. Rales at one apex point to tuberculosis.

PLEURAL FRICTION-SOUND.—When inflamed, as in *acute pleurisy*, the two surfaces of the pleura are roughened, due to the swelling and dilatation of the capillaries, or to the transudation of fluid or lymph. When the two surfaces are rubbing together under these circumstances, a sound is created to which the term friction is applied. It is heard at the end of inspiration and may continue during expiration. The sound is localized usually at the seat of pain; it is *superficial*, being heard near the ear, and is not modified by cough or by full breathing, except occasionally by the latter, when it is repeated; it may be *increased* by the *pressure of the stethoscope*; it is a *fixed* sound in that it does not disappear until effusion takes place; it may reappear when the fluid

has been given to it. It is characteristic of hydropneumothorax and pyopneumothorax, although not present in all cases of these diseases. The sound may be audible at a distance. Metallic tinkling can usually be heard at the same time.

UNVERICHT'S WATER-WHISTLE NOISE.—A bubbling, metallic noise heard in pyopneumothorax with a patent pulmonary fistula, due to the bubbling of air from the fistula up through the fluid in inspiration.

Auscultation of the Voice.—When the ear or stethoscope is applied to the surface of the chest and the patient is asked to speak, the vibrations of the air in the trachea and bronchial tubes, set up by the act of phonation and transmitted to the chest-wall become audible. The sound is known as the *vocal resonance*. It is a sign which goes hand-in-hand with *vocal* or *tactile fremitus*, both being modified by the same condition. Sometimes one is increased and not the other, without there being any evident reason for it.

METHOD OF PROCEDURE.—The patient should repeat in a fixed monotone either the words one, two, three, or ninety-nine, whichever the auscultator is used to hearing. Symmetrical portions of the two sides of the chest must be examined successively, during loud and whispered speaking, the latter frequently showing differences in the two sides which are not appreciable with loud fremitus.

VOCAL RESONANCE IN HEALTH.—Vocal resonance varies in health conjointly with the fremitus. The sound normally is purring or buzzing. It is heard more pronouncedly at the right apex than at the left, in persons with thin chest-walls, and in individuals in whom the voice is low in pitch and strong. It is lessened therefore in females and children. It diminishes the farther away the ear gets from the larynx, and hence is feeblar at the bases.

VOCAL RESONANCE IN DISEASE.—*Increased Vocal Resonance.*—The degree of increased vocal resonance depends upon the intensity or extent of the cause. When slightly above normal, it is referred to as a slight increase. This usually is due to a slight infiltration or a solidification covered with a thickened pleura. The vocal resonance is also increased over cavities.

Bronchophony is the name given the vibratory, blurring sound that is heard when the voice is transmitted with increased loudness to the ear. This may be heard in health over the trachea and large bronchi anteriorly or over the bronchi posteriorly. When heard over the vesicular structures of the lung, it indicates that the vibrations are transmitted to the ear by some better conducting material, usually a solidified lung. In all cases of solidification bronchophony may exist; sometimes the sound is even more pronounced than when heard over the trachea.

Vocal Resonance Diminished.—Vocal resonance is diminished or absent when anything cuts off the supply of air or intercepts the vibrations at the part the observer is auscultating. Vocal resonance is absent over the area supplied by a bronchus which is occluded as by external

pressure, such as that of an aneurism. Diminution or absence of vocal resonance is marked in cases of pleural effusion (serum, blood, pus, or air) or of thickened pleura, the vibrations being impeded because of the difference of conducting material. The degree of diminution depends upon the amount of effusion.

Modifications of Vocal Resonance.—Certain modifications of vocal resonance may occur.

Pectoriloquy is the transmission of syllabic speech. The voice may be as distinctly transmitted as if the patient were speaking into the mouth of the stethoscope. If the patient speaks slowly, the words may be distinctly made out. It is often more striking when the patient whispers. The term *whispering pectoriloquy* is then applied to it. It is most exaggerated over a cavity communicating with a large bronchus, but is frequently heard over small areas of infiltration as the apex of the seat of early tuberculosis.

At the uppermost limit of the pleural effusions, at which point the layer of fluid is thin, the resonance is transmitted in a modified form. It is tremulous and bleating in character, and is known as *egophony*, because it resembles the bleat of a goat. It is heard especially at the angle of the scapula, or below it in cases of moderate effusion. The vocal resonance may have a *metallic* character in pneumothorax when there is free communication with the bronchus.

CHAPTER XXII

PHYSICAL DIAGNOSIS OF DISEASES WITHIN THE ABDOMEN

THE ABDOMEN

THE abdomen is divided arbitrarily into regions to enable us to locate the various organs in health and in disease.

A satisfactory method is to divide the abdomen into quadrants by imaginary lines, one drawn perpendicularly through the umbilicus, from the tip of ensiform to the symphysis pubis, the other drawn horizontally and also passing through the umbilicus. The *right upper quadrant* contains the right lobe of the liver, the gall-bladder, the hepatic flexure and part of the transverse colon, a portion of the pancreas, the pylorus near the median line, and, deeper, the upper half of the kidney. The *left upper quadrant* contains the left lobe of the liver, the stomach, part of the transverse colon and the splenic flexure, the pancreas, the upper portion of the kidney, and the spleen. The *right lower quadrant* contains the cecum, the ascending colon, the appendix vermiformis, the right tube and ovary, portions of the bladder and uterus, and above, at the end of full inspiration, the lower portion of the kidney. The *left lower quadrant* contains the corresponding tube, ovary, and portions of the bladder and uterus, the descending colon, and the sigmoid flexure. It does not usually contain the lower portion of the left kidney, which is one-half inch or more higher than the right. (Holden.) About the centre and extending to the periphery on all sides are the small and large intestines. The abdomen may also be divided into regions by two horizontal and two perpendicular lines. The imaginary horizontal lines are drawn one at the level of the tenth costal cartilages, the infracostal line, and the other at the level of the anterior superior iliac spines, the iliac line. The perpendicular lines are continuations of the midclavicular lines to the middle of Poupart's ligaments. Nine regions are thus made. The upper three are known as the right and left hypochondriac and the epigastric, the middle three as the right and left lumbar and umbilical, and the lower three as the right and left iliac and the hypogastric.

Inspection.—We note the appearance of the abdominal walls, the movements of the abdomen, its shape and size (general enlargement), and the presence of local enlargements.

The Abdominal Walls.—A glance suffices to determine the thickness of the abdominal walls. *Thin* walls are due to deficiency of adipose

tissue, and of muscular structure, either associated with general atrophy or sometimes caused by intra-abdominal pressure. Frequent pregnancies, previous ascites, or antecedent growths (ovarian tumors) lead to atrophy of the muscles; the recti separate and a hernia-like protrusion of abdominal contents results. Furthermore, a conical projection of the lower median portion of the abdomen is brought about, especially if ascites is present. *Thick* walls are due to edema or to increase in fat.

Color.—The abdomen in general partakes of the hue of the skin; around the umbilicus it is darker, and in Addison's disease a distinct areola is often present. The median line, from the umbilicus to the pubis, darkens in pregnancy. This "brown line," as it is called, is sometimes seen in men.

FIG. 103



Pendulous abdomen and lineæ albicantes.

Markings.—In first pregnancies and when there is marked ascites, less frequently in the presence of obesity and tumors, *striæ* are produced in the skin where the tension has been greatest. In pregnancy they form sinuous lines upon the lower lateral portions of the abdominal wall and upon the upper inner portions of the thighs. When first developed, they are reddish, but subsequently by a process of fading become more glistening and white than the rest of the skin. They are known as *lineæ albicantes* (Fig. 103). They may be seen after typhoid fever if the distention has been excessive.

The Umbilicus.—*Pouting* occurs, in pregnancy after the sixth month with portal obstruction, with hernia, and when there is ascites or abdominal distention. Not frequently the walls around the umb

infiltrated with carcinoma secondary to cancer of the stomach and by excising a small portion of the tissue and examining it under the microscopes a correct diagnosis of the internal disease can often be made. Retraction of the navel occurs in stout people.

Veins.—Enlargement of the superficial veins is a common accompaniment of cirrhosis of the liver, pyelophlebitis, or of any condition causing obstruction to the free circulation of the blood in the inferior vena cava. To complete the collateral circulation the veins of the abdomen may anastomose with the mammary veins above or the epigastric veins below, and when the venous distention around the umbilicus is excessive, a characteristic radiating tumor, known as *caput Medusæ*, results.

Movements.—(See The Lungs—Dyspnea.)—The *movements* of the abdomen are of respiratory, vascular, gastric, and intestinal origin.

Respiratory Movements.—When the abdomen is enlarged or contains a tumor in its upper half, the normal movement, synchronous with respiration, is restricted. In paralysis of the diaphragm the abdomen retracts during inspiration and the normal respiratory movement is reversed. When the paralysis is limited to one side, the inspiratory collapse is unilateral. In laryngeal and tracheal obstruction inspiratory retraction is noteworthy. Respiration causes the liver to rise and fall; in persons with thin walls its shadow can be seen to descend with inspiration. A tumor connects with the liver and an enlarged gall-bladder will move synchronously with respiration. Other tumors, unless adherent to the liver, are fixed, excepting pyloric growths, which show moderate respiratory mobility. A rare exception is found in a right kidney movable with respiration.

Vascular Movements.—These are noted in the median line and usually in the upper half of the abdomen. In moderately thin subjects *epigastric pulsation* (*q. v.*) may be directly caused by the aorta. If the pulsation extends some distance to the right or left of the median line, an aneurism may be suspected; or a growth, such as carcinoma of the stomach, overlying the aorta and transmitting its pulsations. Aneurism of the celiac axis gives rise to a movement near the umbilicus and to the right or left of the median line.

Pulsation of the liver, which is of vascular origin and therefore synchronous with cardiac pulsation, is seen in the hepatic area in tricuspid insufficiency.

Gastric and Intestinal Movements.—*Peristaltic movement*, whether of the stomach or of the large or small intestine, may be visible through the abdominal walls. In gastric dilatation and gastroptosis the waves may be seen in rhythmical succession passing from left to right in the centre of the abdomen. When due to movements of the large intestine, the waves follow the course of the canal, while those which emanate from the small intestine are confined to the region around the umbilicus. Visible peristalsis when of gastric origin indicates obstruction at the pylorus; intestinal peristalsis is seen when the lumen of the bowel is

obstructed. The movements may be excited by striking the abdomen with a towel wrung out of cold water, or with the hand.

Shape.—In general enlargement of the abdomen the shape is uniform. In very fat subjects and in women with relaxed abdominal walls the abdomen may be *pendulous*. (See Fig. 103.) In ascites the tissue over the umbilicus may protrude and form a localized prominence in women whose abdominal walls have previously been relaxed. Abdominal enlargements due to ascites sometimes assume a peculiar cone-shape, the base corresponding to the plane of the abdomen, the apex protruding below the umbilicus. Local enlargements, such as morbid growths or changes in the size of viscera, produce irregularities in the surface corresponding in position to the internal lesion. The shape varies momentarily in hysterical distention. In wasting disease of the viscera, as cancer of the retroperitoneal glands, the enlargement is replaced by retraction during the later stages, causing undue prominence of the affected viscera.

Palpation and Percussion.—Palpation and percussion in diseases of the abdomen may be discussed together.

Position of Patient.—Generally the best position is the recumbent one because the abdominal muscles are relaxed; when the abdominal muscles need to be particularly relaxed, the head and shoulders should be partly elevated and the knees drawn up. A good plan to secure muscular relaxation when palpating the liver and spleen is to have the patient sit on a chair with the body leaning forward and the thighs flexed. The knee-chest or hand-and-knee position is of advantage at times in distinguishing between an aneurism and pulsating abdominal tumor, as the latter falls away from the vessels in these positions. A tumor surrounded by coils of intestine may become more distinctly palpable.

Method.—The examining hand should be warm, as the sudden application of cold throws the abdominal muscles into involuntary contraction.

When it is desired to explore deeply, the patient should be instructed to breathe with the mouth open, and the examining hand pressed firmly in during expiration, and held there, if need be, during several long respirations. The palm of the hand should be laid upon the surface; after the muscles are relaxed, the flexed fingers may be used to palpate. The same procedure is adopted when it is desirable to get the percussion note of a body lying deep in the abdomen—that is, the finger is pressed firmly and deeply in and then percussed; in this way any superficial resonance due to overlying intestine is largely eliminated.

When palpating to determine the lower edge of the *liver* or *spleen*, the palmar surfaces of the fingers are pressed into the abdomen at different levels from below upward until the edge of the organ is felt. When the liver is in its normal position, the edge of the right lobe extends to the margin of the ribs, and may be found by pressing the fingers in as described and having the patient take a long breath.

By *palpation* the information obtained by inspection is confirmed; the character of the abdominal walls and of any enlargement that may be present is determined; the precise location of pain is ascertained; the condition at the hernia rings and the movability of tumors are investigated. By passing the hand gently over the surface of the abdomen, any marked *unevenness* such as is produced by umbilical or inguinal hernia, by *striæ*, by large tumors of the pylorus, by cancerous nodules, or by hydatid cyst of the liver, is at once detected. The degree of *tension* of the abdominal walls is easily appreciated. It is decreased or relaxed after pregnancy, dropsy, and the removal of large tumors. It is increased, although not uniformly, whenever the abdomen is very much enlarged. *Rigidity* of the abdominal walls may be the only sign of acute peritonitis, and is quite common when the disease is local. Local contractions point to inflammation underneath. Tuberculous peritonitis is characterized by distention with board-like rigidity or preternatural hardness. *Peritoneal friction* is most frequently felt over the liver, and occurs chiefly in chronic peritonitis.

General Enlargement of the Abdomen.—The abdomen differs very much in size in different persons, depending upon the thickness of the fat in the abdominal walls and omentum. In general, the belly is more protuberant in infants and children than in adults. Enlargement of the belly is only one of the features, though frequently the most pronounced evidence of obesity, whereas in enlargements of the abdomen from *tumors* and *ascites* there is usually a marked contrast between the size of the abdomen and that of the rest of the body.

During the last month or so of *pregnancy* the abdominal enlargement is general, especially when the woman has previously borne children.

Enlargement due to *accumulation of gas* within the bowels is general and may attain a very high degree, lending the abdomen a uniformly arched appearance like that of a barrel. The diaphragm may be forced upward so as to interfere seriously with respiration and heart action. In debilitated children the enlargement due to flatulence is associated with flaccidity of the abdominal walls, causing lateral and central enlargement.

A moderate degree of distention may be the result of eating certain articles of food, such as turnips or beans. Excessive accumulations of gas are observed in typhoid fever; peritonitis, operative and non-operative; intestinal paresis or obstruction; and in stenosis of the colon or rectum from any cause. Excessive distention is also common in hysteria.

Dilatation of the Colon.—In children the enlargement of the abdomen may be general. The dilatation may be temporary, as in constipation with obstruction, or in rare instances permanent, when the distention of the abdomen gradually becomes enormous.

Enlargement of the abdomen simulating ascites may be due to retro-peritoneal and peritoneal *lipomas*. Fluctuation even may be detected, but repeated puncture fails to reveal fluid.

Other causes of general abdominal enlargement are *hydatid cyst*, *cancer of the bowel or peritoneum*, *fecal accumulation*, and *diseases of the liver and gall-bladder*. Enlargement of the latter organs may give rise to only a local swelling in the right upper quadrant; but when they attain very large dimensions, as happens not infrequently in cancer, amyloid disease, and hydatid cyst of the liver, inspection may show only general enlargement, with small prominences corresponding to cancerous nodules or small cysts.

Splenic enlargement attains its greatest development in leukemia and in chronic malarial poisoning. It is often visible as a general enlargement of the belly, but may also produce a more marked prominence over the lower left ribs and the corresponding lumbar region.

In *diseases of the kidneys* associated with great enlargement a prominence is usually visible in the flanks and lumbar region corresponding to the kidney involved unless there is considerable emaciation; anteriorly the enlargement, if any be visible, usually appears to be general.

Enlargements of the abdomen beginning in the lower quadrants and becoming general secondarily are usually of pelvic origin. The most common are those due to *pregnancy*, *retroperitoneal sarcoma*, *ovarian or parovarian cysts*, *uterine fibroids* and *fibrocysts*, and *abscess or peritoneal effusion* as in chronic peritonitis. As a greatly distended bladder may give rise to confusion, it is a good rule to pass a catheter and make sure that the viscus is empty before proceeding further with the examination.

Local Enlargements or Tumors of the Abdomen.—An enlargement in the *region below the sternum* or epigastrium usually indicates distention or dilatation of the stomach or a tumor at the pylorus, which is almost always cancerous. Epigastric prominence is also seen in large eaters. It is not uncommon, however, to find here a *cancerous nodule* projecting from the surface of the liver or a *hydatid cyst* of the same organ. *Aneurism*, *cancer of the large intestine*, and a morbid growth in the *left lobe of the liver* are among the possible causes of enlargement in this region, in which, or to the left of the median line and near the level of the umbilicus, *effusions into the lesser peritoneal cavity* are also found. Much more rarely a swelling in the epigastrium is caused by a tumor of the pancreas, such as cyst, abscess, or cancer, or by sclerosis of that organ.

A *rigid rectus muscle* is capable of simulating a tumor. In hysterical subjects rigidity of the abdominal muscles with tympanites sometimes gives rise to a swelling known as "phantom tumor." Such swellings are less constant in shape and character than genuine tumors, and, although dull on percussion, appear more superficial; they sometimes disappear under friction with the hand, and invariably under full anesthesia; when in addition the stigmas of hysteria are present all doubt is at once removed. These phantom tumors are, as Fritz has pointed out, often really cases of dilatation of the colon.

Enlargements in the *right upper quadrant* or right hypochondrium are most frequently due to disease of the liver and gall-bladder. Less

frequently a much *enlarged* or *cystic kidney* causes swelling in this region.

Primary malignant disease of the suprarenal bodies—a rare affection—is often accompanied by swelling of the upper abdomen.

Enlargement in the *right lower quadrant* or right iliac region occurs with affections of the *cecum* and *appendix*, as appendicitis, typhlitis, and perityphlitis, with or without pus formation, fecal accumulation and fecal abscess, carcinoma, stricture of the ileocecal valve, intussusception, and with *ovarian* and *tubal disease*, such as ovarian tumor, cyst of the broad ligament, pelvic abscess—usually of tubal origin—and extra-uterine pregnancy.

Other conditions to be thought of in this region are acute and chronic *tuberculous peritonitis* and an *enlarged* or *movable kidney*. In these conditions the most exact information in regard to physical characteristics is obtained by means of palpation and percussion, which, with the clinical history, enable the physician to distinguish one from the other.

When the *left upper quadrant* or left hypochondrium is the seat of enlargement one of the following conditions should be suspected: dilatation or carcinoma of the stomach; enlargement of the spleen (*q. v.*); movable kidney or a renal tumor; and effusion in the lesser peritoneal cavity.

The enlargement may also be due to *fecal accumulation* in the left transverse and descending colon.

An interesting cause of swelling in this region and in the lumbar region is *perigastric* or *subdiaphragmatic abscess* (*q. v.*).

Enlargements in the *left lower quadrant* or left iliac region in women are usually due to ovarian tumors, pelvic abscess, pelvic hematocele, or uterine fibroid, the diagnostic points of which have been referred to under enlargements in the right iliac region. When these conditions can be excluded, an enlargement in this region may indicate fecal accumulation or tumor—usually cancerous—in the sigmoid flexure or descending colon, volvulus, tuberculous peritonitis, or enlargement or displacement of the kidney or spleen (*q. v.*). Fecal abscess also may occur here, and the tumor of intussusception may be found on the left side.

Enlargement *about the centre of the abdomen* or umbilical region may be due to umbilical hernia, to gastropexia, or a floating kidney, spleen, or liver, to cancer of the stomach, liver, or intestine, or sarcoma of the retroperitoneal glands, to hydatid disease of the liver or a tumor of the gall-bladder, or finally to tuberculous disease of the omentum or mesenteric glands (*tabes mesenterica*).

Enlargement *above the pubis* or in the hypogastric region is due most frequently to pregnancy, fibroid tumor or fibrocyst of the uterus, or to distention of the bladder. In *acute tuberculous peritonitis* a diffuse swelling of gradual development sometimes appears in this region. Enlargement above the pubis, associated with flattening of the upper half of the abdomen and abnormal distinctness of the lesser curvature, also occur in *dilatation of the stomach* and *gastropexia*.

Enlargements in the *flanks and lumbar regions* accompany malignant tumors of the kidneys, hydronephrosis and pyonephrosis, perinephric abscess, and large renal cysts; although, as a rule, renal enlargements do not produce any visible change in the back. On the *left side* an enlargement may also be due to perigastric or subdiaphragmatic abscess, or to an enlarged or dislocated spleen. On the *right side* the cause may be an enlargement, as from hydatid cyst of the liver, or a retroperitoneal sarcoma.

Diminution in Size of the Abdomen—Retraction.—The size of the abdomen is diminished in wasting diseases, and in diseases that either interfere with the ingestion or assimilation of food or destroy the appetite. This class comprises cancer of the esophagus and stomach, chronic lead-poisoning, anorexia nervosa, chronic diarrhea, and tuberculosis of childhood. Wasting of the subcutaneous and omental fat and atrophy of the abdominal organs cause the abdomen to become concave or scaphoid.

Auscultation.—Abdominal auscultation is only occasionally of value in physical examinations. The absence of the normal peristaltic sounds is a diagnostic sign of importance in peritonitis, and is the result of intestinal paresis. In mechanical intestinal obstruction the sounds are usually exaggerated above the obstruction in the effort of peristalsis to push by the obstruction. Crepitant friction is occasionally heard over the liver or spleen in peritonitis. Over a gravid uterus can be heard fetal heart sounds and the uterus soufflé. A bruit can at times be noticed when there is an abdominal aneurism. A venous hum is also heard occasionally in the region of the umbilicus, in cirrhosis of the liver due to the enlargement of the umbilical or para-umbilical veins. The sounds heard over the stomach are described in the physical examination of that organ.

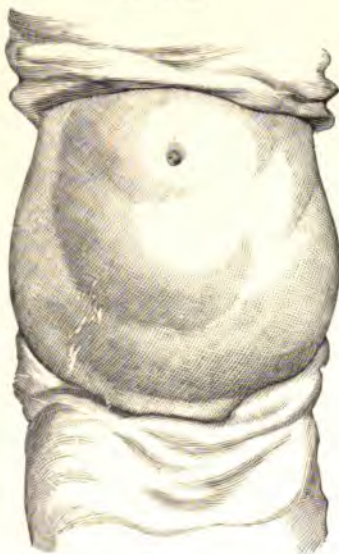
THE STOMACH

Inspection.—Direct inspection of the epigastric region often affords much positive information. When there is much loss of abdominal fat and the stomach is well distended, its outlines can sometimes be traced with the eye, and the position and size of the viscus may be outlined by observing the shadow corresponding to the lower curvature, which moves with respiration. A shadow corresponding to the lesser curvature is also seen in gastropotosis. The best position for the examiner is behind and above the patient's head while the latter is lying down. If the lower curvature can be traced considerably below the navel, the stomach is almost certainly dilated, and if at the same time there is a prominent swelling in the pyloric region the dilatation is probably due to cancer of the pylorus. A marked groove extending from the umbilicus to the ribs, about or to the left of the nipple-line, is a case of dilatation with gastropotosis. It marks the position

curvature. The lower border is also marked by a groove extending in a curve from the pubis toward the first groove.

Peristaltic waves may be seen to move spontaneously from left to right, or after tapping the region, or applying an ether spray or ice-water. When the pylorus is obstructed, antiperistaltic waves may also be seen.

FIG. 104



Stomach inflated, showing gastropptosis.

Distention.—Distention of the stomach with carbon dioxide or, safer and better, with air by means of a hand-bulb syringe (see inflation), frequently brings the outlines of tumors of the pylorus plainly into view, while at the same time any tumor lying behind the stomach becomes less distinct, and false tumors due to spasm of the gastric muscular coat vanish. Distention also helps to map out the entire organ, to separate it from surrounding viscera, and to estimate its size and position. It therefore affords a means of distinguishing gastropptosis from dilatation.

Gastrodiaphany or Transillumination of the Stomach.—Einhorn has succeeded in transilluminating the stomach with an Edison lamp fastened to a soft-rubber tube. The wires to the battery are carried through the tube. After the stomach contents have been removed, the patient is to take one or two glassfuls of water, and the apparatus after lubrication is then inserted. The examination must be made in a dark room. By means of gastrodiaphany the approximate position and size of the stomach and the presence of tumors of the anterior wall are determined. The results are not strictly accurate, however, as the

intestines when empty are also illuminated. The form and size of the stomach are not so readily brought out as the topographical relation of tumors of the stomach and those in the vicinity of that organ. Gastro-diaphany is of service in some cases to distinguish dilatation from gastropptosis.

Direct Gastroscopy.—The esophagus and stomach may be directly examined by means of a long thin metal tube with a small electric light and a reflecting mirror on the distal end, the gastroscope. It is claimed that through this tube, ulcerations, erosions, thickenings, and inflammatory conditions can be recognized anywhere in the stomach. This direct method of examination of the stomach is hardly available for ordinary clinical procedures, as it requires a certain amount of technical skill to pass the tube, and a general anesthetic is usually necessary.

Röntgen Rays.—The outline and movements of the stomach may be observed by the use of the fluoroscope, provided the patient has been previously given an emulsion of bismuth subnitrate.

Palpation.—Palpation of the stomach is closely associated with auscultation, inasmuch as the former also elicits sounds (succussion, gurgling) which are helpful in diagnosis. The hand must be placed flat upon the abdomen and pressure made by flexing the last phalanges. To make deep palpation, gradually increasing pressure with a rotary movement must be employed. It may be of advantage to palpate in the knee-elbow position, so that deeply seated tumors, if movable, may fall forward against the abdominal walls. Palpation, however, elicits information independently of auscultation, chiefly in conditions of disease.

Epigastric pulsation is common in anemia, in nervous dyspepsia, in valvular disease of the heart, particularly tricuspid regurgitation, which produces a liver pulse, and in rare cases of aneurism of the abdominal aorta.

Increased resistance may be due to hypertrophy of the muscular coat which coexists with distention of the stomach. A shrunken condition of the stomach with increased resistance may be due to a diffused carcinoma of the walls of the stomach; or, rarely, to the so-called "fibroid stomach"—atrophy and thickening of the walls from chronic gastritis.

Increased resistance limited to the pylorus is found in *carcinoma*. The same effect produced by a tense right rectus muscle must be excluded.

Position of Gastric Tumors.—Cancers of the pylorus are situated usually between the xiphoid cartilage and the umbilicus, frequently a little to the right of the median line; but they may be found below umbilicus and, exceptionally, still lower down. Adhesions to neighboring organs commonly prevent the tumor from being moved. If it has formed adhesions to the liver or diaphragm, it moves with respiration, and even when there are no adhesions, pyloric tumors may move a little with respiration, and may often be displaced so

inches laterally by manipulation or change of posture. Tumors of the lesser curvature show decided respiratory movement. As a rule, tumors due to gastric cancer are small, hard, and irregular, and gradually increase in size.

Non-malignant tumors are found in rare instances, as well as tumors due to adhesions around old ulcers and to puckered scars. The latter are distinguished from cancerous tumors not by the physical examination, but by their duration and clinical history. Sometimes a fibroid pylorus may be felt as a firm, cylindrical mass about the size of the terminal phalanx of the thumb.

Tenderness.—Tenderness is elicited by palpation in gastritis, in neuroses, especially the hyperacid forms, in ulcer, and in cancer. In gastritis and neuroses the tenderness is usually diffuse and is not constant; in cancer the tenderness is usually limited to the seat of the tumor, but is not so marked or so sharply localized as in ulcer. In ulcer tenderness is rarely absent; even when there is no pain it is very decided, and sometimes is so sharply localized that the tender spot can be covered with the tip of the finger.

Percussion.—**Position of the Stomach.**—The stomach is a distensible organ, and does not occupy a fixed position. It is depressed by downward pressure of the diaphragm in deep inspiration, by emphysema, left pleural effusions, enlargements of the liver and spleen, and tight lacing—it is elevated by any causes that greatly distend the bowels or peritoneal cavity—tympanites, peritoneal effusions, tumors, and the like.

When at rest its shape is that of a tube, the upper two-thirds of which lies in the left upper quadrant and runs almost vertically downward from the cardia. The lower third curves toward the pylorus and lies almost horizontally. After food is taken the stomach is distended and its position changed, the organ being rotated anteriorly from below, the greater curvature rising and looking more forward, while the anterior surface has a more upward presentation.

The *cardiac end* of the stomach is fixed to the diaphragm by peritoneal attachments; the cardiac orifice lies behind the sternal insertion of the left seventh rib. The *pylorus*, on the contrary, is freely movable and its position may vary considerably. It usually lies slightly to the right of the midline about half way between the umbilicus and xiphoid. Variations in the position of the greater curvature of the stomach are also compatible with health. It may extend normally as low as the umbilicus. In children the position is usually higher, while in adults over fifty it frequently lies below the navel. The *upper border* of the stomach, in the left midclavicular line, lies between the fifth interspace and the sixth rib, occasionally in the fourth interspace or at the seventh rib.

Traube has called special attention to the left lower portion of the thorax which projects over the stomach, the "semilunar space." The upper limit is a crescentic line starting from the sternum in the sixth

interspace and extending in a curved line, corresponding approximately to the curve of the rib, to the axillary line. It is known as "Traube's line." In health this space gives a tympanitic note, unless the stomach or transverse colon is full or the omentum very fatty. In left pleural effusion it is dull. (See Diseases of Lungs.)

Area of Stomach Tympany.—Though the tympanitic percussion note over the empty stomach, the so-called "stomach tympany," is said to have certain qualities which distinguish it from tympany of the rest of the abdomen, in actual practice it is usually impossible to outline the stomach by ordinary percussion. However, if the patient has just taken some water or food into the stomach the lower border can usually be demonstrated. Auscultatory percussion is frequently valuable, especially if the stomach is inflated or contains water, and is of particular value in differentiating a tumor of the pylorus from a tumor in the same position unconnected with the stomach.

Auscultation.—By means of auscultation it can be determined whether or not there is obstruction at the cardiac orifice. On listening over the esophagus with the stethoscope, while the patient is swallowing a liquid, a spurting sound is heard, followed in from five to ten or twelve seconds by a second sound, which marks the escape of the fluid from the cardiac orifice of the esophagus into the stomach, the so-called "deglutition murmur." When there is obstruction at the cardiac orifice, the second sound may be delayed as long as a minute.

When the stomach is partly filled with fluid, a *succussion* or *splashing* sound can be produced by moving the patient quickly from side to side, or by quickly compressing the stomach and allowing it to rebound immediately. Such compression may be made alternately, first in the neighborhood of the fundus of the stomach and then in the region of the umbilicus. Both hands should be employed. The ear need not be applied to the body while the movements are made, as the sound is audible at a distance of several inches. Such sounds are abnormal if they are heard more than three hours after a light, or six hours after a full meal. Dilatation is very probable if the splashing sound is heard below the navel in a fasting stomach.

Inflation of the Stomach.—The most accurate method of determining the size, shape, and position of the stomach is by inflation. Positive information of these factors by actual distention of the viscus with air, supplemented by other methods of physical examination, is readily obtained, and the most practicable as well as the most effective means of accomplishing this object is by simple inflation through a stomach-tube with an ordinary atomizer bulb. The carbon dioxide method has the sole advantage of enabling one to dispense with the introduction of a stomach-tube, and so many distinct disadvantages and dangers that its use cannot be countenanced. With ordinary inflation, if the air is pumped in rapidly at first, spasm is induced at the pylorus and prevents the premature escape of the distending medium into the small intestine. (Pepper and Stengel.) The stomach should first be

emptied in the usual manner, after which an atomizer bulb is fitted to the stomach-tube, without removing the latter, and the distention carried to a point just short of causing discomfort. Owing to the lax condition of the abdominal walls, the outline of the stomach is usually at once visible, or it can be determined by palpation, by percussion, or by auscultatory percussion. The following sign may be sufficient: "when the bulb of the syringe is compressed, forcing the air through the tube, a peculiar metallic ring can be heard with the stethoscope over the stomach. When the bell of the instrument is moved along beyond the stomach limits, the sound instantly loses its metallic character." The same authority advises that the air be allowed to escape before the tube is withdrawn, in order to prevent the distress which often accompanies the escape of air from the stomach, probably on account of a spasm at the cardiac orifice.

When gastropotosis is present, the greater curvature is found well below the level of the umbilicus, and in extreme cases may lie in the pelvis. Dilatation will show a marked increase in the size of the stomach. Visible peristalsis is frequently noted only after inflation and tumors of the anterior wall and pylorus may appear, or become more prominent if previously noted; while tumors posteriorly, if previously palpable, disappear. The counter-indications of this method are such as would apply to the passage of a stomach-tube at any time.

THE INTESTINES

Inspection.—Local and general enlargements of the abdomen have been discussed in the preceding pages. Movements due to increased peristalsis of the intestines are seen in obstruction. The intestine above the point of obstruction may swell into a well-defined tumor, which becomes hard, and is dull tympanitic on percussion.

Palpation.—Tenderness, peristalsis, peritoneal friction, the bubbling of gas through a constriction of the bowel, and tumors are recognized by palpation. It is often necessary to place the patient on all-fours or in the knee-chest position.

Percussion.—The normal note is tympanitic. Local areas of dullness may be due to intestinal tumor. Light percussion should be employed. A dull tympany indicates a solid mass surrounded by distended intestines. The outline of the large intestine can be ascertained by filling it with water.

Auscultation.—Normally, peristaltic sounds are heard over the intestines. These are absent if there is paresis of the bowel from peritonitis or from other causes. Exaggeration in frequency and intensity of the sounds take place when there is increased peristalsis, as in enteritis, after a cathartic, and in the early stages of mechanical intestinal obstruction above the site of the obstruction.

THE LIVER

Topographical Anatomy.—(See Plates IV and V.) The right lobe of the liver is applied to the concavity formed by the lower lobe of the right lung, being separated from it by the diaphragm. The thin lower edge of the right lung overlaps the liver at its upper part, but the greater portion of the anterior surface of the right lobe of the liver is in contact with the ribs. The under surface of the liver is in relation with the stomach, transverse colon, duodenum, right kidney, and right suprarenal capsule.

A needle thrust into the right side, in the midaxillary region, between the six and seventh ribs, would traverse the lung, and then go through the diaphragm at its central attachment, into the liver. The lower border of the liver extends in the median line one-third of the distance from the tip of the xiphoid cartilage to the umbilicus. In the right midclavicular line it extends to the lower border of the ribs; and in the midaxillary to the tenth rib. The upper border is opposite the upper border of the sixth rib in the midclavicular line, and extends horizontally to the axilla to the ninth rib behind.

The attachment of the liver permits of a certain amount of movement. Hence, the liver is depressed by deep inspiration, emphysema of the lungs, or right pleural effusion. When the patient lies upon his left side, the left lobe of the liver rises higher and the right extends lower; and *vice versa*, when the patient lies upon the right side, the liver turning upon the suspensory ligament as an axis. (Gerhardt.)

Inspection.—Inspection is not of very great assistance in the diagnosis of diseases of the liver. Frequently there is a *swelling* or *tumor* in the right upper quadrant, which may or may not be produced by an enlargement of the liver, but which should direct attention to that organ. Such a swelling may be observed in amyloid disease, hydatid tumor, cancer, abscess, and less frequently in fatty liver.

Palpation.—The lower border of the liver can be palpated in thin subjects, and often the gall-bladder when it is greatly enlarged, although difficult when the abdomen is distended with gas. Palpation must be made with the tips of the fingers, pressing them firmly inward along the margin of the ribs, at the same time procuring relaxation of the abdominal muscles by having the patient take a full breath, and having the legs drawn up and the shoulders elevated. The pressure should be made in the interval following the acts of inspiration. By care and patience the fingers can be pushed deeply inward and be made to feel the border of the liver, even in health. Care must be taken not to cause contraction of the right rectus muscle, for when rigid it may simulate tumor or enlargement of the liver. The left lobe of the liver, below the ensiform cartilage, extends almost half-way to the umbilicus. Here it is most accessible to palpation. By palpation we learn the extent of movement of the liver during respiration. The liver descends

and ascends with inspiration and expiration. This movability distinguishes it from fixed organs.

Glénard's method, or *procédé du pouce*, sometimes enables the examiner to feel the edge of the liver when ordinary methods of palpation fail to reveal it. The method consists essentially in thrusting the liver forward with the fingers of the left hand, while with the thumb in abduction the edge of the liver is felt for during deep inspiration, the right hand meanwhile forcing the intestines out of the way. (See Fig. 105.)

FIG. 105



Glénard's procédé du pouce.

The liver is uniformly enlarged and palpable in fatty and amyloid disease, congestion, multiple abscess, acute hepatitis, phosphorus-poisoning, leukemia, rickets, congenital syphilis, obstruction of the bile ducts, and cirrhosis. Irregular palpable enlargements are found in single abscess, cancer, hydatid cyst on the anterior or lower surface, and syphilis (gummas). The liver may also be palpable, though not enlarged when pushed downward by large pleural effusions, emphysema, subdiaphragmatic abscess, etc. Enlargement of one lobe of the liver may be the result of any causes of an irregular enlargement, or it may

be the result of constriction of the liver from tight lacing, or a "Reidel's lobe" may be present.

Floating liver is diagnosticated by feeling in the lower, most frequently the right, portion of the belly a large tumor which may, however, easily be confounded with tumors of other organs. It can be distinguished as the liver (1) by recognizing the notch; (2) by the presence of a tympanitic note in the normal hepatic region, as loops of intestine lie between the diaphragm and liver; (3) by the excessive movability of the tumor; (4) by the fact that it is possible to replace the liver; and (5) by its size and consistency. It occurs almost exclusively in women, possibly as the result of a congenital lengthening of the suspensory ligament, although more likely from relaxed abdominal walls. It may be confounded with ovarian cyst, appendicitis with tumor, and movable right kidney with hydronephrosis.

Palpation of the liver may discover a *friction* from perihepatitis, and *pain or tenderness* from that cause or from cancer or abscess. *Pulsation* of the liver may be a transmitted impulse from the abdominal aorta or a venous pulse, such as occurs also in the jugulars from tricuspid regurgitation.

Gall-bladder.—When the gall-bladder has a certain degree of fulness, it may, according to Gerhardt, be not only felt in healthy persons, if the stomach and bowel are empty, as a smooth, round, fluctuating tumor at the lower border of the liver, but be even visible and be outlined by percussion. If a line is drawn from the right acromion process to the umbilicus, it will bisect the gall-bladder at the point where it passes over the margin of the ribs. The fundus is situated below the edge of the liver, at about the ninth costal cartilage, just outside the edge of the right rectus muscle. Palpation is easy when, owing to closure of the cystic duct, the gall-bladder is distended with bile or inflammatory exudate, or enlarged by thickening of its walls or an accumulation of gall-stones. A pear-shaped tumor is then felt which, if not adherent to the border of the liver, is movable with it. In simple stasis and in purulent inflammation the tumor is tense and elastic; in inflammatory or carcinomatous thickening of the wall, dense and irregular. Calculi can often be recognized by their form or hardness, or by the sound made by rubbing them together.

Percussion.—**Limits of Liver Dulness in Health.**—The upper border of the liver dulness is found by percussing from above downward toward the liver, beginning anteriorly at the third interspace, on the lateral surface of the chest at the fourth, and posteriorly at the angle of the scapula. In health the upper border is found anteriorly at the fifth interspace, in the axilla at the sixth, and in the back at the ninth interspace; and the liver dulness should extend downward to the margin of the ribs. In the aged the lower border falls short of this position by at least an inch, and in deep chested persons the entire liver dulness may be not more than two inches in front. The width of the liver dulness is about 4 inches in the right midclavicular line, 6 inches in

the midaxillary line, and 3 inches in the midscapular line. In children the lower border of the liver is normally lower than in adults on account of the relatively greater size of the viscus. For the same reason the upper border is at a higher level.

Diminution in size can be determined by percussion only, and is usually best recognized in the anterior and lateral regions. Diminution in size is due to simple or acute yellow *atrophy* of the liver or to *cirrhosis*. It must not be confounded with the apparent diminution that takes place in emphysema, or that which occurs from distention of the bowels with flatus, as in peritonitis. Absence of hepatic dulness may occur when there is gas in the peritoneal cavity. When there is much distention of the intestines by gas, the anterior and lateral hepatic areas may be tympanitic.

Enlargement of the Liver.—In order to detect enlargement of the liver, inspection, palpation, and percussion—both superficial and deep—are employed; palpatory percussion is sometimes of great advantage. Any marked increase in the hepatic dulness beyond the normal limits (see page 336) usually means a corresponding increase in the size of the organ. The enlargement may be uniform and the shape of the liver dulness normal, or the outline may be irregular, or again the enlargement may be limited to one lobe. If the enlargement is irregular, the liver dulness may begin at a higher point in the anterior than in the lateral region, or may extend beyond the margin of the ribs in a limited area.

When the enlargement is limited to the left lobe, it is revealed by increase in the dulness from the xiphoid cartilage downward as far as the umbilicus. The entire mid abdomen to the navel may be filled by the enlarged liver. The causes of *uniform enlargement* and *enlargement of one lobe of the liver* have been discussed. (See page 337.)

Irregularity in the shape of the liver dulness occurs in cancer, in abscess, in hydatid disease, in gumma, in congenital malformations, and in corset liver. Although in abscess or hydatid disease enlargement *downward* is more common, it may be directly upward, the lower border of the liver occupying the normal position. When enlargement of the liver extends *upward* it is due to a cyst, abscess, or gumma in the convex surface of the right lobe.

DIAGNOSIS.—Enlargement of the liver from disease must be distinguished from enlargement of organs in contiguity with the liver, from structures usually containing air which have become solid or non-resonant and from congenital or acquired malformations. It is well to bear in mind the conditions which *simulate enlargements of the liver*. Of these we have:

1. *Pleural Effusion.*—The difficulty in distinguishing enlargement of the liver from a pleural effusion arises because the hepatic dulness is continuous with that caused by the presence of fluid in the pleural sac. In pleural effusion, however, there is uniform bulging of the affected side; the liver is not movable, and chest expansion is lessened.

The upper border of dulness may be movable if the effusion is not large, and the line of dulness, S-shaped—that is, high behind and high in front. If the effusion is large, the upper limit of dulness is horizontal. Sometimes an enlargement of the liver may give rise to secondary pleural effusion, so that too often, after finding pleural effusion, the size of the liver is not estimated.

2. *Lobar Pneumonia*.—The history and clinical course and physical signs will immediately differentiate the two conditions.

3. *Pericardial effusion and dilatation of the heart* are said to simulate enlargement of the liver. The history of the case, the origin and mode of development of the symptoms, and the physical signs of cardiac disease, point to the true nature of the lesion.

4. *Subdiaphragmatic Abscess*.—The history of the case is generally essential to a diagnosis. The accumulation between the liver and diaphragm causes the latter to be pushed downward. It is very difficult to distinguish the true from the false enlargement in these instances. *Aspiration* may help in the diagnosis.

5. *Abnormal Condition of the Abdominal Parietes*.—Increased tension or spasm of the recti muscles, giving rise to phantom tumors of the abdomen, simulate enlargement of the liver in hysteria. Anesthesia must often be employed to disperse the swelling.

6. *Disease of the spinal column* causes dislocation, on account of which the liver may apparently be increased in size.

7. *Congenital Malformation*.—Congenital malformations may be suspected in the absence of any symptoms of hepatic disease or of conditions that may cause other forms of spurious enlargement. Moreover, the increased dulness will have existed from early life.

8. *Tight Lacing*.—This may displace the liver upward or downward, according to the direction of the pressure. It may also, by exerting lateral compression, bring more of the liver into contact with the anterior abdominal wall; and finally, if the constriction is due to pressure of a strap or tight cord, a portion of the liver may be more or less detached and appear as a movable tumor.

9. *Enlargement of the abdominal contents* may cause spurious enlargement of the liver. In the same way increased abdominal pressure from ascites or tympanites causes the liver to rise higher than normal. (a) *The accumulation of feces in the colon*. This causes extension of the liver dulness downward, on account of which it may be thought that the patient has liver disease. A purgative must be given. (b) *An ovarian cyst*. (c) *Ascites*. Exclusion of ascites is sometimes difficult if the ascites is loculated and situated in the hepatic region. (d) *Tumors of the omentum*, chiefly tuberculous, may occupy such relation to the liver as to increase the dulness downward. The history, the occurrence of the omental tumor, with symptoms of tuberculosis, may aid in determining the condition. (e) *In tumors of the kidney*, which simulate enlarged liver, it is found that the edge of the liver cannot well be felt; but the fingers can usually be inserted between the

and the upper part of the renal tumor. The renal tumor, however, is not fixed. It is rounded on every side, and has the well-known shape of a kidney. (f) Enlargements of the liver must be distinguished from *pancreatic cyst*, or *effusion into the lesser peritoneal cavity*. This can usually be accomplished with ease, except in hydatid disease of the left lobe near the suspensory ligament. In effusion into the lesser peritoneal cavity the tumor occupies the left upper quadrant, and may extend downward to the level of the umbilicus. It causes dislocation of the heart, so that the apex is as high as the third interspace and beyond the midclavicular line. It is accompanied by an increase in the dulness posteriorly, so that the upper limit may extend to the angle of the left scapula. Puncture may furnish the necessary information.

Aspiration.—We are rarely warranted in determining the nature of an obscure enlargement of the liver or of the gall-bladder by aspiration. In abscess pus is found; in hydatid disease, the characteristic fluid may be withdrawn. In a case of local enlargement, the apex of the swelling should be aspirated. If puncture is performed near the upper border, the needle should be thrust downward; if the near lower border, upward. The left lobe should be aspirated with care, in order to avoid entering the stomach.

Auscultation.—By auscultation we may detect a *friction sound* in perihepatitis; a *grating* or *rubbing* during palpation when the gall-bladder contains calculi; a continuous murmur in tricuspid regurgitation.

THE SPLEEN

Topography of the Spleen (Plate V).—The spleen lies in the left upper quadrant, beneath and in contact with the diaphragm above, and with the tail of the pancreas, the cardiac end of the stomach, and the suprarenal gland below. It extends from the upper border of the ninth to the lower border of the eleventh rib, and from the mid-axillary line backward and upward toward the spine.

Palpation.—As the spleen lies entirely behind the ribs, it does not, under normal conditions, admit of palpation; but when it is *enlarged* it becomes accessible to palpation in proportion to the degree of enlargement and of the relaxation of the abdominal walls. It *moves with respiration* and cannot be said to be enlarged unless the edge of the organ is palpable at the end of a deep inspiration, even if there is increased dulness in the lower axillary region. An enlarged spleen usually retains its normal *shape*, and the direction of the enlargement is *downward* and *inward*. When the enlargement is moderate, the smooth, blunt, rounded anterior surface and sharp edge of the spleen can be felt at the margin of the ribs during deep inspiration; when the enlargement is great, as in *leukemia*, the organ can be grasped with both hands and its hilus clearly mapped out. The same thing can be done in the rare instances of *floating spleen*, but here a knee-chest

position will favor successful palpation. The posterior border of an enlarged spleen can usually be made out by passing the hand backward over the resisting organ; in children this border is always readily found. In fact, the spleen is always more readily palpable in children than in adults; it is also more movable, and therefore can be brought forward more easily to the median by bimanual palpation.

FIG. 106



Feeling for the edge of the spleen.

In *splenic leukemia* the spleen may be larger after a meal, yield a creaking fremitus on palpation and a murmur on auscultation, and may even pulsate. The spleen may also *diminish* in size after diarrhea or free hemorrhage.

Percussion.—The spleen yields a *dull sound* on percussion. Posteriorly and below, the splenic dulness merges into that of the lumbar region and kidney. The upper posterior portion is hidden behind the diaphragm and overlapping lung, and hence is not accessible to percussion. Practically, therefore, the *normal splenic dulness* extends between the ninth and eleventh ribs in the middle and posterior axillary lines, the spleen being there in contact with the ribs. (See Plate V.)

In percussion of the spleen the patient should lie on his right side. Beginning from above downward, the interspaces are percussed gently until pulmonary resonance is replaced by dulness; then proceeding from the gastric area toward the axilla until tympany yields to dulness.

In the same way, percussing from below upward, the line is reached where the intestinal tympany gives way to dulness.

Splenic dulness may be encroached upon by tympany due to the stomach or colon distended with gas; or its dulness may appear increased through distention of the stomach and colon with solid matter, a left pleural effusion, or basal pneumonia. The spleen may also be pressed upward by ascites or by a large abdominal tumor, so that its normal dulness is much lessened.

THE KIDNEYS

Topography of the Kidneys (Plate IV, Fig. 2).—The kidneys are situated in the right and left lumbar regions respectively, the left being a little higher than the right. They extend from the eleventh rib, or twelfth dorsal vertebra, to the third lumbar vertebra. The left kidney is in contact above the spleen, and the right with the liver.

The kidneys are enveloped in fat; their distance from the anterior surface of the abdomen renders them inaccessible to percussion from that direction, and the thick dorsal and lumbar tissues coupled with the relation of the kidneys with the spleen and liver, which give a dull note on percussion, make it difficult to outline the kidneys from behind.

Palpation.—Palpation of the normal kidney is difficult. It can only be bimanual. Place the fingers of one hand below the last rib outside of the lumbar muscles—erector spinæ—and apply the other below the ribs in front. Firm, persistent pressure with the abdominal muscles relaxed, especially in thin subjects, will often enable the normal kidney on the right side to be felt when the patient takes a deep breath.

Palpation of the kidney becomes easy when it is either enlarged or displaced, and at times the kidney can be grasped between the two hands, its size estimated, and its physical characteristics as regards hardness, softness, fluctuation, and mobility determined. The fact that the tumor moves a little with respiration aids in its detection; and if it is unusually movable the edge of the hand can be slipped above its upper end by turning edgewise that border of the hand which is adjacent to the ribs. A renal tumor is usually two or three inches to either side of the median line, a little above the level of the umbilicus.

A very favorable position for palpating movable kidneys is that assumed by standing and leaning forward over a chair, with the trunk supported by the hands resting on the seat of the chair. In this position the abdominal muscles are relaxed and the kidneys fall forward.

In the diagnosis of renal tumors, in general, it should be borne in mind that they are slightly *movable* with *respiration* unless adherent, as in malignant disease, abscess, and cysts. Unless too large they preserve their *reniform* shape, and press in front of them the ascending or descending *colon*. The position of the colon should therefore always be ascertained, and to this end it may be necessary to inflate it.

Percussion.—The best results are obtained by having the patient lie face downward, and placing a cushion under the belly so as to make the lumbar regions a little more prominent. Strong percussion is required, and an artificial plessor and pleximeter are to be preferred. Percussion should be conducted with a view to marking the angle which the liver dulness and splenic dulness make with that of the kidney on the right and left side respectively. The kidneys extend below the lower lines of liver and splenic dulness, and laterally for a width not greater than four inches. The difficulties in the way of outlining the kidneys by percussion are greatly increased in persons with much flesh, in muscular subjects when there is ascites, and it is practically impossible under such circumstances to be sure of the boundaries of the kidneys. The colon must be emptied to yield trustworthy results.

CHAPTER XXIII

THE RÖNTGEN RAY IN MEDICAL DIAGNOSIS

THE employment of the Röntgen ray in medicine is an extremely important adjuvant to other methods of diagnosis. Its use, however, is outside of the realm of the ordinary practitioner, not only because of the primary expense of the apparatus but because the proper taking and correct interpretation of an x-ray plate requires special skill and considerable experience which can only be attained by constant practice in this branch of medicine. It follows, therefore, that a diagnosis from a plate or through the use of the fluoroscope should be left to specialists in this line of work. The internist, however, should be acquainted with those conditions in which the x-ray examination is of value and should be prepared to make use of this diagnostic method in questionable and obscure cases. In the following sections the various conditions in which the Röntgen ray or the fluoroscope are of diagnostic worth will be briefly considered.

The Head.—The head is the most difficult region that the skiagrapher has to deal with, as this cavity is enclosed by a bony envelope with numerous irregularities, particularly in the base; sutures separate the various bones; there are grooves for the lodgement of vessels; density and thickness of the bony encasement change at every point; large venous sinuses are present; the brain is extremely vascular; the external ear throws a distinct shadow, and there are many large air cavities with thin walls. All these irregularities and anatomical structures tend to make the shadow of the head very complex. The results of skiagraphy of brain tumors, cysts, abscess, or blood-clot are, on the whole, of doubtful value for diagnostic purposes. A few men have been able to obtain shadows of suspected tumors and other lesions, and to confirm their findings by autopsy. Such tumors as sarcomas, tuberculomas, gummas, and fibromas, when definitely localized, have been demonstrated in the skiagraph by skilful operators. Tumors of the pituitary body are recognized by alterations in the sella turcica. Blood-clots being rather opaque, when they are near the surface, as in extradural hemorrhage, are shown with greater ease and yield fairly clear shadows. A thick-walled abscess within the vault may be demonstrated and abscess or new growths in the accessory sinuses are usually clearly shown. Cystic conditions are represented by a diminution in the normal density of the shadow of the area, and the skiagraph may be of value in determining the diagnosis between the cyst, a clot, and a tumor. The nearer the lesion is to the side of the head and to the plate, the denser

will be the shadow and the clearer its outline. The fluoroscope is of little or no value in this part of the body.

The Neck.—The size, shape, and position of an aneurism or a tumor of this region can often be demonstrated, as can cervical ribs.

The Thorax.—In this part of the body the x-ray is of great usefulness; not only are the lungs, heart, and great vessels accurately shown, but also mediastinal new growths, abscesses, and enlarged peribronchial glands. The movements and position of the diaphragm are best studied by the fluoroscope. Alterations in the position and movements of the normal bilaterally dome-shaped diaphragm may be the result of almost any pathological condition of the lungs or pleural cavity. The diaphragm will be unilaterally depressed by a hydrothorax, pneumothorax, or pyothorax or a new growth in the lower lungs or pleura or by a pericardial effusion; bilaterally depressed by intrapulmonary conditions such as asthma or emphysema or by fluids in both pleural cavities; or pulled down irregularly by inflammatory conditions with adhesion in the abdominal cavity. It may be elevated by fibrous bands and adhesion, as a result of inflammatory conditions in the pleura, notably tuberculous, or pushed up by ascites or new growths, abscesses, and cysts of the near-by structures or organs in the abdominal cavity. In these conditions the movements of the diaphragm are usually limited or absolutely immobile on one side or both sides, depending upon the lesion. Limitation of motion of the diaphragm occurs also on the affected side in conditions not actually involving the muscle, as tuberculosis or pneumonia of an upper lobe of a lung.

Heart.—The position, shape, size, and actions of the heart may be studied, preferably by the fluoroscope. Pathological conditions of the pericardium are usually clearly seen. By means of the orthodiagraph, an instrument used to determine the exact size of an organ by deflection of the x-rays so as to be made parallel, a graphic representation of the heart may be made and preserved for subsequent study and record.

Aorta.—Dilatation and aneurism of the aorta are clearly shown.

Lungs and Pleura.—Examination in disease should note *abnormal dark shadows, area of abnormal brightness, and alterations in the height, shape, and movements of the diaphragm.* Shadows found in place of a normal penetration may be indicative of such conditions as infiltrations and solidifications of phthisis, pneumonia, malignant growths of the lung, pleura, or mediastinum, gangrene, infarct, compressed lung, echinococcus cyst, empyema, thickened pleura, pleural effusions, etc. Emphysema and pneumothorax may allow of greater transmission of the rays and give greater brightness than normal.

Tuberculosis.—In *tuberculosis* the x-ray is of questionable value in the diagnosis of an early infiltration of an apex or elsewhere. Certainly it should be depended upon only in conjunction with other symptoms and physical signs of such a lesion. In the latter stages of the disease it is of value in studying further pathological changes that may

take place, in determining the presence of complication, such as empyema, and in arriving at a diagnosis in the presence of other pulmonary conditions which obscure the physical findings.

Pneumonia.—Here the x-ray is of special value in recognizing deep-seated lesions, central unresolved pneumonias, empyema, and pneumonia in the presence of other pulmonary diseases.

Pleural Effusion.—Valuable in recognizing small effusions of which the physical signs are questionable or the extent of secondary inflammatory changes.

Esophagus.—Dilatations, diverticula, and structures are readily demonstrable. New growths are seen with difficulty.

Abdomen.—Until recently the x-ray was used but little in the diagnosis of intra-abdominal conditions. The brilliant results that have been achieved in recent years in the study of the physiology and the pathology of the gastro-intestinal tract by means of the fluoroscope have opened up splendid fields of usefulness for this instrument in the diagnosis of obscure and difficult lesions in the stomach and intestine.

Stomach.—Abnormalities in the size, shape, and position of the stomach are readily demonstrated. Tumors, ulcers, cicatrices, and contractions of the stomach wall and pylorus are shown by the skiagraph; at times they are usually more satisfactorily studied by the fluoroscope which shows alterations in the peristaltic waves when they reach such conditions which may not actually cast a shadow. The motor power of the stomach is readily determined and the presence of pyloric obstructions is clearly shown. The secretory power of the stomach has also been studied by the fluoroscope.

Intestines.—Abnormalities and displacements of the cecum, colon, and sigmoid are readily diagnosed. Kinks, diverticula, strictures, dilatations, and obstructions within the lumen of the intestine can be seen with the fluoroscope. Tumors of the wall are demonstrable at times and the character of the intestinal peristalsis can be studied.

Liver and Gall-bladder.—Very little information of value can be obtained by examination of the liver, except perhaps as to its size, position, and shape when abnormal. Gall-stones unless containing much lime salts, which is rare, are not observed.

Kidneys.—The size, shape, and position are demonstrable. Perinephritic abscesses cast a distinct shadow. Urinary calculi in the pelvis or ureter are practically always shown when present. Injection of the ureter with collargol through a ureteral catheter shows beautifully dilatations of the pelvis of the kidney from a hydro- or pyonephrosis.

The Extremities.—Diseases of the bones and joints, atheromatous conditions and aneurisms of the peripheral vessels are easy to demonstrate by the x-ray.

SECTION V

LABORATORY DIAGNOSIS

CHAPTER XXIV

THE BLOOD

Normal Blood.—The blood consists of corpuscles and plasma. The formed elements are (1) red-blood cells (erythrocytes); (2) leukocytes; and (3) platelets. In former times much stress was laid upon the physical character of the blood drawn in bulk. For accuracy in diagnosis, however, reliance must be placed upon instruments of precision, the microscope, the hemoglobinometer, and the hemocytometer. By these we determine (1) the size and shape of the red cells; (2) the morphological characteristics of the white cells; (3) the number of red cells; (4) the number of the white cells; (5) the presence of new elements, as nucleated red cells and myelocytes; (6) the presence of parasites; (7) the amount of hemoglobin.

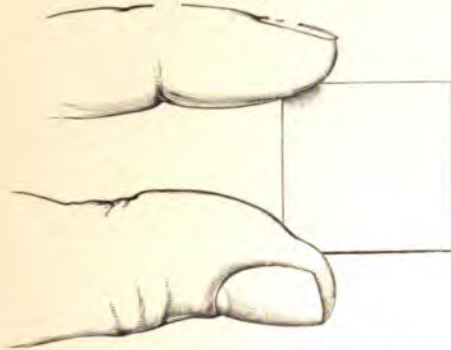
Technique.—A drop of blood may be taken from the lobe of the ear or the finger tip. The surface should be thoroughly cleansed with alcohol and dried carefully. The puncture should be made forcibly and quickly, in order that the blood may ooze freely. A bayonet-pointed instrument, devised for the purpose, should be used, or the nib of a new steel pen, one-half of which has been broken off, answers fully as well. As soon as the blood flows freely without pressure, the apex of a drop may be touched by the cover-glass, which has been previously cleansed in alcohol and dried upon a clean handkerchief. The cover-glass should not touch the skin, and as soon as it is touched by the blood it should be placed face downward, somewhat diagonally, upon a corresponding cover-glass. The blood will then spread evenly over the surface without pressure upon the cover-glasses. As soon as spreading ceases, slide the glasses one off the other, but do not lift them apart. In a similar way, using a large drop of blood, two slides may be employed instead of cover-slips, or the drop may be spread over a slide by a fine tissue paper or by the edge of another slide drawn sharply across the surface. At the same time the blood for other necessary tests is collected.

Blood collected in this way may be examined at once, vaseline being put around the margins of the slip to prevent the entrance of air, or be put aside for staining and future examination.

EXAMINATION OF FRESH BLOOD.—By the examination of fresh blood we learn of the presence of parasites, the degree of coloring of the red cells, and their shape and size, and the presence of blood platelets. In a well-prepared specimen the rouleaux, "money roll," formations are avoided except as the blood masses toward the edges of the cover-slip.

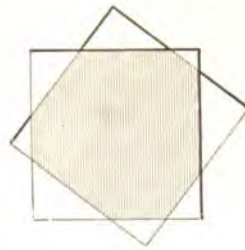
Here may be mentioned the hemokonium granules, or blood-dust (Müller), the small dancing coccus-like refractive bodies seen in greater or less abundance in all blood; their significance is unknown, and they are not readily recognized in stained specimens.

FIG. 107



Proper method of holding a cover-glass.
(Cabot.)

FIG. 108



Illustrating the position of a
cover-glass during the spreading
of blood films. (Cabot.)

Examination of Stained Preparations.—Cover-slip preparations of the blood are stained particularly to determine the different kinds and numbers of the white corpuscles, the presence of blast cells and of granular degeneration. They are first "fixed" then stained.

Fixation.—Fixation is usually done during the process of staining by the alcohol contained in the alcoholic stain. If a non-alcoholic stain is to be employed, the slips are readily and thoroughly fixed by immersion in strong alcohol for ten minutes and then washed. Fixation by heat at a temperature of 110° C. applied for two minutes is also satisfactory.

Staining.—Eosin-methylene Blue.—A simple method of staining blood films is by the use of 1 per cent. eosin solution in 70 per cent. alcohol, followed by 0.5 per cent. aqueous methylene blue. The film is stained in the eosin for thirty to sixty seconds, washed and stained with methylene blue for a slightly shorter time. The protoplasm of the red cells and polynuclears stain pink, the nuclei and the protoplasm of the lymphocytes blue, and the eosinophilic granules red, the basophilic deep blue, and the neutrophilic bluish red.

Romanowsky Stain (Leishman's modification).—This stain can be purchased in powder form or in small soloids¹ which are dissolved in 15 c.c. of pure methyl alcohol so that a really good stain can be prepared with a minimum of trouble in a few minutes; it is of particular value in office and general work.

Technique.—Place enough of the stain on the cover-slip to cover it. Leave this on for thirty seconds so that the alcohol will fix the blood. Now add water, drop by drop, until a metallic scum appears on the surface of the stain. The stain is allowed to remain for two or three minutes, after which the film is washed until it appears pinkish, and is then dried. As the alcohol evaporates rapidly it is best to cover the film while staining with a Petri dish. Greater accuracy is secured if the same amount of stain and water is used in each film and if the exact time it takes to stain satisfactorily is ascertained by staining several films for different lengths of time. The red cells should appear pinkish, the leukocytic, nuclei, and blast cells dark blue or purple, the protoplasm of the lymphocytes light blue, the eosinophilic granules bright red, and other granules dark blue or lilac.

Wright's stain may be made up in the same way; the technique of the staining is the same; and the tinctorial characteristics are the same as the Romanowsky method.

Giemsa's Method.—This stain can also be bought in soloids and the dye made up in a few minutes. The blood film is fixed in methyl alcohol for a minute and covered for five minutes with a mixture of the stain in the proportion of 14 drops to 10 c.c. of distilled water. The various stained elements appear essentially as with Leishman's modification.

Jenner's Stain.—The powdered stain can be purchased as with the other stains, or a 1.25 per cent. aqueous solution of eosin and a 1 per cent. aqueous solution of acid-free methylene blue are mixed in equal parts and allowed to stand uncovered for forty-eight hours. The resulting precipitate is washed with water, the filtrate collected, dried and powdered. A 0.5 per cent. solution in absolute methyl alcohol is then made up. The blood films are stained and fixed, at the same time, for three minutes. The red cells appear, when examined microscopically, as a grayish-pink tint; the nuclei of blast cells and leukocytes blue; the neutrophilic granules reddish purple; the eosinophilic granules brilliant red; and the basophilic granules a deep violet.

The Red Corpuscles or Erythrocytes.—The ordinary red-blood cells measure $\frac{1}{3200}$ inch (7μ to 8μ). In an adult man the red cells number from 5,000,000 to 5,500,000 to the cubic millimeter; in an adult woman the number is usually less, being from 4,500,000 to 5,000,000. In thinly spread films the red cells are recognized by their color and shape. They vary from 6μ to 9μ in diameter. The lighter colored

¹ This stain and the following stains are prepared and sold in dry form by Grüber or Burroughs-Welch Co.

centre, due to the biconcavity of the corpuscle, sometimes causes confusion. It must be remembered, too, that the corpuscles readily become crenated. A slight molecular movement is sometimes also seen, which must not be confounded with the ameboid movements in drying cells, or with the rapid motion of malarial pigment.

Variations.—Variations in size and shape are indications of disease. In anemia the red cells may be larger or smaller than normal (anisocytosis). Large cells are known as macrocytes, small cells as microcytes. Cells that are irregular in shape are known as poikilocytes. They may be oval, pointed, angular or reniform.

Nucleated Red Corpuscles or Erythroblasts.—These contain one or more nuclei. The stroma takes the acid stain and the nucleus the pure basic stain. They are divided in accordance with their size and the depth of the color of the nuclei, into three varieties:

1. *The Normoblast.*—This is the size of a normal red-blood corpuscle. The one or more nuclei are deeply bluish black and homogeneous and occupy one-fourth to three-fourths of the whole corpuscle. The normoblast is the parent cell of the red blood corpuscle, and is found in anemia, particularly after severe hemorrhage or in disease of the bone-marrow.

2. *The Megaloblast.*—This cell is larger than a normal red-blood corpuscle, the nucleus is bluish green rather than black, and not compact and homogeneous, showing a well-marked nuclear net-work. The megaloblast is found in the marrow of the embryo and in severe anemias.

3. *The Microblast.*—This is a small blast cell rarely seen.

Basophilia.—In this condition the protoplasm of some of the erythrocytes stains more strongly with the basic than the acid stain. Three forms of basophilia are recognized.

1. Homogeneous basophilia (polychromatophilia). The entire cell is of a homogeneous pale blue color.

2. Granular basophilia (granular degeneration). The cell contains numerous fine bluish granules, the intervening protoplasm being pinkish.

3. Stippling or malarial dotting. Minute intracellular bluish-black granules dot the pinkish protoplasm.

The first two forms are evidences of degeneration of the cells and consequently are found at the same time in most anemias. However, the granular degeneration, which is particularly characteristic of lead and other mineral poisoning, may be pronounced while polychromatophilia is slight or absent. Malarial dotting is seen in the tertian, but rarely in the estivo-autumnal type of malaria.

Blood Counting.—The hemocytometer or blood counter most frequently used in this country is that of Thoma-Zeiss. It consists of a heavy glass slide (*a*), in the middle of which is a cell (*B*) exactly $\frac{1}{10}$ mm. in depth. The cell is limited at the periphery by a circular gutter to prevent fluid placed upon the cell from flowing in between the slide and the cover-glass. The floor of the cell is ruled into squares whose

until Newton's color ring appears between the cover-slip and outer portion of the slide, and then the slip is allowed to stand from three to five minutes until the corpuscles have settled to the bottom of the cell.

The cell is ruled into 400 small squares, groups of 16 squares being separated by double lines. The surface of a small square is $\frac{1}{400}$ sq. mm., and the depth of the cell being $\frac{1}{10}$ mm., the space overlying each square is $\frac{1}{4000}$ c.mm. As each small square equals $\frac{1}{4000}$ c.mm., the number of cells in 4000 of these little squares would equal the number of cells in each c.mm. As such counting would be too tedious, the number of erythrocytes in 1 c.mm. of blood is estimated by counting the number of erythrocytes in a certain number of small squares and then multiplying the number of cells counted by the product of the sum of the number of small squares equal to 1 c.mm. (4000) multiplied by the dilution (200) divided by the number of small squares counted. To simplify the technique, erythrocytes are counted in five groups of sixteen small squares, *i. e.*, 80 small squares. This will give the following equation: $4000 \times 200 \div 80 = 10,000$. Therefore, multiply the number of cells counted in 80 small squares by 10,000 or simply add four ciphers to the total number of erythrocytes counted and the product gives the number of erythrocytes in 1 c.mm. The groups of sixteen small squares should be selected in different parts of the field, that is, choose a group in each corner and in the centre of the slide. The erythrocytes touching the lines bordering the upper and right side of a square should be counted in the number of cells in that square, those touching the lower and left side should be omitted, in order to prevent counting the same cells twice or omitting any cells, and in order to have a standard whereby it can be uniformly determined what cells should be counted in the square and what cells should be considered outside of the square.

As diluting fluids, a 1 per cent. solution of common salt may be employed. Toisson's fluid (distilled water, 160 c.c.; glycerin, 30 c.c.; sodium sulphate, 8 grams; sodium chloride, 1 gram; methyl-violet, 0.025 gram) or Hayem's solution (mercuric chloride, 0.5 gram; sodium sulphate, 5 grams; sodium chloride, 1 gram aq. dest. 200 c.c.) are preferable, however.

In the estimation of the white blood cells the pipette made by Zeiss is employed. In this instrument the blood is diluted twenty times by a solution of a 1.3 per cent. acetic acid solution. By means of this solution red cells are dissolved and the nuclei of the white cells are rendered distinct and easy of recognition. The ordinary Thoma-Zeiss slide is employed. To obtain accurate results, four entire fields of 400 squares should be counted. The number of leukocytes in each of the four fields are added together and divided by four, giving the average number of leukocytes in an average field of 400 squares. Then the number of leukocytes in 1 c.mm. of blood equal 4000×20 (the dilution) \div the average number of squares counted (400) \times average number of leukocytes in four fields, which is equivalent to multiplying

the average leukocytes by 200 or dividing the total number counted by 2 and adding 2 ciphers.

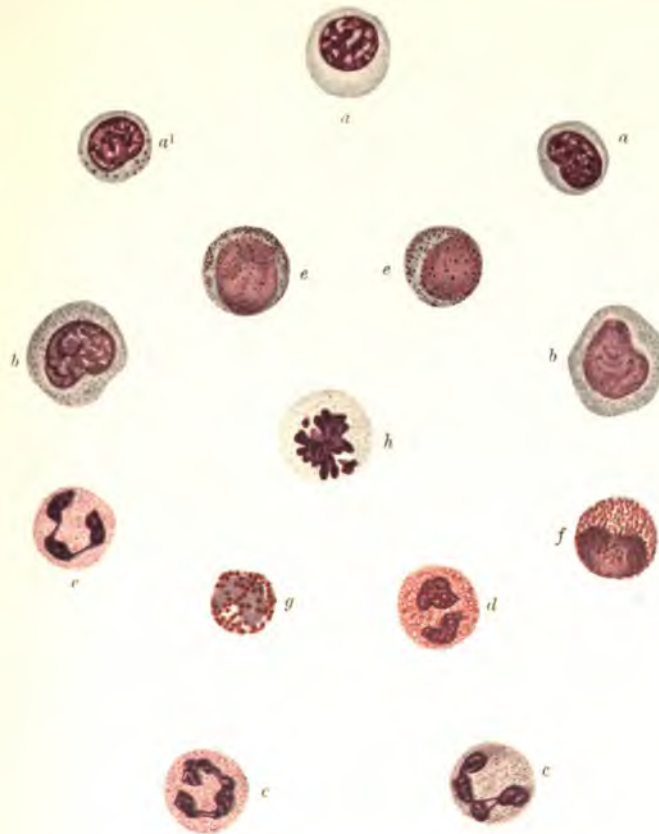
Number of Red Blood Corpuscles in Health.—The normal number of red cells, as already stated, is approximately 5,000,000 per c.mm. They may be reduced to 500,000. A reduction below 3,000,000 indicates grave anemia. When the reduction is below 1,500,000 the anemia is said to be pernicious or malignant. It must be remembered that the red cells are temporarily reduced during menstruation and lactation. At puberty there is also a reduction. When the blood is concentrated by profuse sweating or exhaustive diarrhea, the number of red cells is increased, while on the other hand they are lowered when the blood is diluted by large draughts of fluid or by subcutaneous injections of fluid. A cold bath may temporarily concentrate the peripheral blood, and thereby increase the number of cells. Red cells are always decreased in the aged, and are reduced in number after great exertion. They are increased in number after fasting, and diminished after a meal, particularly if much fluid is taken. Oligocythemia is the name applied to a diminution in the number of red-blood cells, from whatever cause. It is usually associated with oligochromemia (deficiency of hemoglobin).

Polycythemia is the name applied to an increase in the number of erythrocytes. It may be relative, the result of concentration of the body fluids as after severe diarrhea, or absolute, as in congenital heart disease, in repeated poisonings by illuminating gas or in erythrocythemia.

The White Corpuscles.—The white or colorless corpuscles are recognized by their absence of color, by their irregular shape and their size, which is larger than that of the red ($\frac{1}{2500}$ inch), and by the ameboid movements which they undergo, particularly when placed in a warm stage. They number from 4000 to 10,000 per c.mm. They are readily recognized by the peculiar affinity which they have for various aniline dyes. They appear in stained specimens as granular nucleated cells. In addition to determining the number by counting, as described in the paragraph which gives the method of counting the red cells, a so-called differential count is made of the stained leukocytes. This count enables us to determine the proportion of the several varieties of leukocytes.

Varieties of Leukocytes.—In the normal blood are found the following varieties of leukocytes: (1) *Lymphocytes*, or small mononuclears, which are cells about the size of a red blood corpuscle, and have a round, large, deeply stained nucleus, surrounded by a narrow rim of non-granular protoplasm. With the Romanowsky stain a few pinkish dots, "azure" granules, can be seen in the protoplasm. (2) *Large mononuclear* leukocytes several times as large as the small mononuclear form. They have a round or oval nucleus, occasionally indented or kidney-shaped (transitional form), with a relatively larger amount of non-granular protoplasm. (3) *Neutrophilic polynuclear* leukocytes: These are usually somewhat smaller than the larger mononuclear variety

PLATE VI



Leukocytes. (Simon.)

a, microlymphocytes; *a'*, same, showing azurophilic granules; *b*, large mononuclear leukocytes; *c*, neutrophilic poly.norphonuclear elements; *d*, adult eosinophile; *e*, neutrophilic myelocytes; *f*, eosinophilic myelocyte; *g*, mast-cell; *h*, karyokinetic normoblast. (Stained with Wright's stain.)



nuclei may be twisted and irregular, and stain deeply. The protoplasm contains granules that are stained by both acid and basophilic dyes. The cells are, therefore, called "neutrophiles." (4) Leukocytes similar to the last form, except that their protoplasm contains large coarse granules that are stained by acid dyes alone. For this reason they are called *eosinophiles*. (5) *Mast cells* (basophiles) appear as a polynuclear or transitional cell with large and small granules which take the basic stains.

The proportion of each variety in the normal blood is: lymphocytes, 23 to 33 per cent.; polymorphonuclears, 60 to 70 per cent.; large mononuclear and transitional forms, 1 to 6 per cent.; eosinophiles, 2 per cent. or less; and basophiles, 0.5 per cent.

Pathological Leukocytes.—Myelocytes.—The occurrence of myelocytes in the blood is pathological. Their well-known occurrence in leukemia and pernicious anemia need not be referred to here. They have been found, however, in a number of infections, particularly in diphtheria, but usually only when there is present a grave form of anemia. Their occasional occurrence is not of great diagnostic value. They are non-ameboid. They are large (15μ in diameter) mononuclear neutrophiles, eosinophiles, or basophiles with large, well-defined, lateral spherical nuclei. Occasionally they are small, when they are recognized by the granules and the very pale large nucleus.

Plasma Cells.—Mononuclear non-granular cells, frequently the size of the large mononuclears, with pale-staining nucleus and deeper staining protoplasm, found in acute and chronic inflammatory conditions.

Differential Counting.—After the specimen is carefully stained it is ready for the differential counting of the white cells, as well as for the determination of the presence of nucleated red cells. The best plan is to pick out a clear field with the low power and then to begin counting at the upper left-hand corner of the field and count across the field to the right-hand corner, using the oil-immersion lens and a movable stage. Then move the slide so that an adjacent field comes into view, when the process is to be repeated. The names of the various forms of leukocytes are placed at the left-hand side of a piece of paper. As the various cells are encountered a dot or a line is placed after that cell until a total of 200 to 300 cells are counted. The percentage of the various forms is then readily estimated. A rough estimation of the number of the erythroblasts can be made at the same time by noting the number seen and comparing that number with the number of leukocytes seen, the number of leukocytes per c.mm. having been previously determined. This last procedure should always be done before making a differential count, as otherwise it would be impossible to tell if an increase or decrease in the several types was absolute or relative.

Leukocytosis.—The term leukocytosis indicates a temporary increase in the number of white blood cells of the same morphological varieties.

1. An *absolute* leukocytosis means an increase in the total

number of leukocytes in 1 c.mm. of blood irrespective of the differential count. A *relative* leukocytosis indicates increase in the percentage of any type of leukocyte without regard to the total number of cells.

An absolute leukocytosis is practically always accompanied by a relative increase either of polynuclear neutrophiles, less frequently of lymphocytes or very rarely of the other forms of leukocytes. Therefore the usual form of leukocytosis is spoken of as a polynuclear neutrophilic leukocytosis, although lymphocytic leukocytosis (lymphocytosis), eosinophilic leukocytosis, etc., may also occur.

A more or less persistent increase in the white cells is frequently spoken of as a hyperleukocytosis, a decrease as a hypoleukocytosis (leukopenia).

Pathological Leukocytosis.—Polynuclear neutrophilic leukocytosis, a relative with usually an absolute increase in these cells, occurs in the following conditions: (1) Acute inflammatory and suppurative disorders, as pleurisy, pericarditis, meningitis, polyarthritis, appendicitis, pyonephrosis, perinephritic abscess, tonsillar and retropharyngeal abscess, acute pancreatitis, cholangitis, etc. (2) Most acute infectious diseases, as varicella, variola, vaccinia, epidemic cerebrospinal meningitis, cholera, typhus fever, lobar pneumonia, glanders, diphtheria, scarlet fever, erysipelas, pyemia and septicemia, and acute articular rheumatism. (3) After hemorrhage. (4) Just before death. (5) After the action of certain drugs as the coal-tar derivatives, quinine, the salicylates, and after inhalations of ether or chloroform. (6) In most cachectic and wasting diseases. On the other hand, in uncomplicated cases of influenza, typhoid fever, tuberculosis when not associated with cavity formation or hyperplasia of lymphatic glands, r  theln, measles, malaria, mumps, pernicious and splenic anemia, and after ingestion of certain drugs (sulphonal, menthol, atropine, tannic acid, etc.), a neutrophilic leukopenia, or relative decrease in the number of neutrophilic leukocytes is present.

Lymphocytosis.—A relative increase of lymphocytes with or without a total increase of leukocytes, is seen in infants, and is commonly marked in rickets and hereditary syphilis. It is found in some forms of scurvy. In adults lymphocytosis occurs in chlorosis and pernicious anemia, and in the secondary anemia of syphilis and typhoid fever. It occurs in hemophilia, in adenitis, and splenic tumors. It is also found at the end of scarlet fever and measles, typhoid fever, gout, in pneumonia with delayed resolution, and in some forms of phthisis (Cabot). Relative and absolute lymphocytosis occurs in lymphatic leukemia and in pertussis.

Eosinophilia.—A relative increase in the eosinophiles, with or without an absolute increase, is seen in the following conditions:

1. Bronchial asthma.
2. Helminthiasis, particularly trichinosis and uncinariasis.
3. Most acute and chronic skin diseases.
4. Malignant tumors, particularly when inva-

5. After febrile conditions causing a polynuclear neutrophilic leukocytosis.
6. Scarlet fever and gonorrhea.
7. Myelocytic leukemia.
8. Uncomplicated ovarian disease.
9. After splenectomy.

Diminution or, more common, absence of eosinophiles takes place notably in pyogenic infections, whether or not associated with a polynuclear leukocytosis.

Myelocytosis.—Normally absent, myelocytes appear in the blood in:

1. Myelocytic leukemia.
2. Severe secondary or primary anemia.
3. Conditions associated with high polynuclear leukocytosis.
4. Malignant disease of the bone-marrow.

Diagnostic Value of Leukocytosis.—The diagnostic value of a differential and leukocytic blood count in diagnosing various diseases is self-evident. It is well to note, moreover, that in most infections the counts are an index to the severity of the infection and the resistance of the individual. In general it may be said that the higher the absolute leukocytosis the better the individual resistance, and the higher the relative neutrophilic increase the greater the severity of the infection though a relative increase of polynuclear neutrophiles to between 80 per cent. to 90 per cent. is approximately normal in infections, so that any increase above these figures indicates a severe infection. For example, in a case showing a slight leukocytosis or a normal leukocytic count with a high (over 90 per cent.) relative polynuclear neutrophilic leukocytosis, the resistance is poor and the infection overwhelming.

Arneth's Differential Count.—Arneth, by careful study of the morphology of the leukocytes, has shown that variation in the nuclei of polynuclear neutrophilic leukocytes is a valuable adjuvant to the study of disease. He divides the leukocytes into two main classes and many subdivisions according to the variations in the nucleus, but for practical purposes they may simply be divided into five classes, according to the number of the lobules of the nucleus, one, two, three, four, or five. Class one and two consist of the young cells having one or two nuclear lobules and are less resistant than the older multinucleated cells. Increase in their proportion and decrease in the proportions of the latter cells, or a deviation to the left shows poor resistance to an infection; deviation to the right, good resistance. Therefore the changes from the normal proportion that takes place in an infection, particularly in pulmonary tuberculosis, are valuable prognostic signs and an aid in showing the result of treatment.

The normal percentage of the several classes is as follows:

	I.	II.	III.	IV.	V.
normal variations . . .	4 to 9	21 to 47	33 to 48	9 to 23	2 to 4 (Simon)
normal percentage . . .	5	35	41	17	2 (Arneth)

graduated tube represents the percentage of hemoglobin contained in the blood tested.

Fleischl's hemoglobinometer consists of (1) a small capillary tube; (2) a well with two compartments and a glass bottom; (3) a small metal stand with a hollow aperture beneath, which is (4) a graduated strip of colored glass, moved by a rack and pinion, and beneath this (5) a reflector. For use, one end of the capillary tube is carefully lowered upon a drop of blood, which immediately fills it. The tube is then washed by a pipette into one of the compartments of the well which

FIG. 111



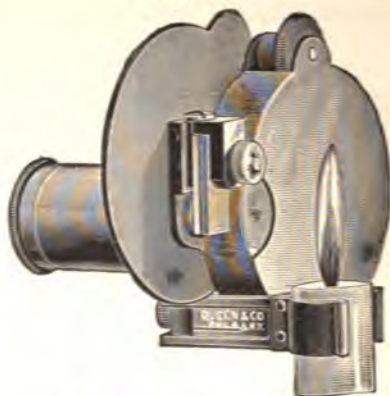
Sahli's hemoglobinometer.

contains some water and thoroughly stirred. The compartments are now equally filled flush with water and the well so placed that yellow light, as from a candle, is reflected through the glass bottom. The colored glass is now moved by the rack and pinion *rapidly* back and forth beneath the water-containing apartment until the intensity of the color in the two compartments is the same, and the percentage is then read off through a small opening behind the well.

Tallquist color scale test for hemoglobin, in which the drop of blood is taken on white filter paper and immediately compared with a scale of colors by daylight, is simple, but the test seems very uncertain.

Dare's hemoglobinometer is compact and reliable, and has the advantage that unadulterated blood may be used. It consists of a large, flat pipette which fills by capillary attraction. This pipette and a color scale are compared directly against a candle flame, being observed through a camera tube; the color of the scale is made to agree with that of the blood, and the percentage of hemoglobin reckoned accordingly.

FIG. 112



Dare's hemoglobinometer.

Color Index.—The ratio between the number of erythrocytes in 1 c.mm. of blood and the percentage of hemoglobin is known as the color index. In a healthy individual with 5,000,000 red cells per c.mm. the normal percentage of hemoglobin should be 100. We then say the color index = 1. If the hemoglobin is diminished, the color index is less than 1, but if the red cells are decreased more than the hemoglobin, the color index is more than 1. The color index is estimated first by reducing the count of the cells to a percentage, *e. g.*, 3,500,000 red cells = 70 per cent.; 4,000,000 = 80 per cent., etc. Second, by dividing this percentage into the hemoglobin percentage. In pernicious anemia the color index is plus 1 and serves perhaps as the most important sign in distinguishing primary from secondary anemia in which the color index is normal or less than 1.

Oligochromemia.—Diminution in the amount of hemoglobin is seen in anemia and usually the reduction is lower than the reduction of the red cells, except in pernicious anemia. In chlorosis the reduction in hemoglobin is very great, and in consequence the color index is lower than in secondary anemias.

Lipemia.—The presence in the blood of fats, usually in the form of small droplets, is easily detected by the microscope. The diagnosis can be confirmed by treating the fresh preparation with a 1 per cent. solution of osmic acid, followed by a weak aqueous solution of eosin. The fat drops will appear black among the faintly stained acid

corpuscles. Lipemia occurs in chronic alcoholism, chronic nephritis, diabetes, tuberculosis, malaria, during starvation, after injuries to the bone-marrow, and in phosphorus-poisoning.

The Glycogen Reaction and Iodophilia.—This reaction consists in the appearance of mahogany-brown granules in the leukocytes and plasma after staining with a mixture of iodine 1 part, potassium iodide 3 parts, water 100 parts, and pulverized acacia in excess. It is present in conditions of purulent or serous exudates and inflammatory processes especially where leukocytosis coexists. It is considered to be due to the presence of glycogen of an amyloid-like substance.

Alkalinity of the Blood.—The technique is too long and difficult and of doubtful import to be available for ordinary clinical procedures. The same may be said of present methods of the estimation of sugar or uric acid, acetone, etc., in the blood, with the added factor that these estimations are usually of little value for diagnostic purposes.

Bile Pigments in the Blood.—Bile pigments are found in the blood in any condition associated with urobilinuria. It can be demonstrated by centrifuging a small specimen of blood. The serum in the upper portion of the tube is normally clear and light yellow in color, but when bilirubin is present it is a bright yellow, gradually becoming greenish on exposure to air.

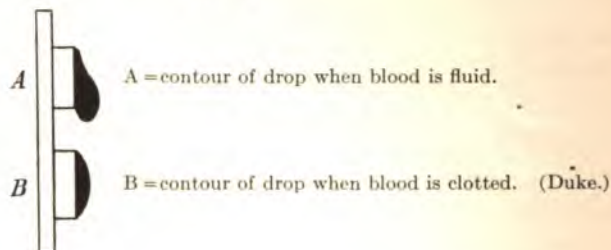
Specific Gravity.—The specific gravity of the blood is in direct relationship to the percentage of hemoglobin except in nephritis, circulatory disturbances, leukemia, and anemia after hemorrhage. Hammerschlag's method of estimation may be employed: mix in a urinometer glass such quantities of chloroform and benzol that the specific gravity is about 1059. Take a drop of blood from the punctured ear with a medicine dropper and blow it into the chloroform-benzol mixture. The blood does not mix, but floats like a red bead. Add chloroform, drop by drop, if the blood sinks to the bottom; add benzol if it rises to the top. After each addition stir the mixture with a glass rod. When the drop remains stationary in the middle of the fluid its specific gravity is the same as that of the fluid. Take the specific gravity of the mixture and you have the specific gravity of the blood. Air should not be blown into the fluid with the blood drop. The following table gives the relations of the hemoglobin to the specific gravity from which an estimate of the specific gravity or hemoglobin can be made:

Specific gravity.	Hemoglobin.
1033 to 1035	25 to 30 per cent.
1035 to 1038	30 to 35 per cent.
1038 to 1040	35 to 40 per cent.
1040 to 1045	40 to 45 per cent.
1045 to 1048	45 to 55 per cent.
1048 to 1050	55 to 65 per cent.
1050 to 1053	65 to 70 per cent.
1053 to 1055	70 to 75 per cent.
1055 to 1057	75 to 85 per cent.
1057 to 1060	85 to 95 per cent.

Coagulation Time is an estimate of the time required for the blood to clot. It is of value particularly before surgical procedures and in various hemorrhagic disorders. A simple method is to put a large drop of blood on a clean glass slide and then tilt it every half minute until the drop form has disappeared when the slide is tilted. The normal coagulation time is from five to eight minutes. In hemophilia and purpura the coagulation time is much delayed. In severe jaundice, particularly the obstructive form, scurvy, typhoid fever, and phosphorus poisoning the coagulation time of the blood is also often considerably delayed.

Duke has devised a simple instrument and method of determining the coagulation time of blood, which he claims is extremely accurate, as the end-point appears sharply; there is always a constant amount of blood used and exposed to the same area of foreign surface; and the temperature is constant. The instrument consists of a glass slide on which are mounted in balsam two glass disks 5 mm. in diameter. To perform the test cover the finger tip lightly with petrolatum and pierce

FIG. 113



through this. Touch the glass disks with a drop of blood sufficient to cover them completely. Insert the slide over a glass containing water at a temperature of 40° C. and cover the top of the glass with a wet cloth of the same temperature. Examine the slide at the end of five minutes and if the coagulation has not occurred the drop hangs down. When coagulation has proceeded to a certain degree the drop form is lost and the blood maintains a spherical contour (Fig. 113). This is considered as the end-point and appears in normal individuals in from six to nine minutes. The two disks may also be used to compare the blood of a patient with the blood of a normal individual.

Parasites in the Blood.—Bacteria.—The principal bacteria demonstrable in the blood are those associated with the infections. Most frequently found, during the course of the infection, are: (1) typhoid, paratyphoid, and colon bacilli; (2) pneumonococci; (3) meningococci; (4) micrococcus melitensis; (5) plague bacilli; (6) streptococci; (7) staphylococci; (8) anthrax bacilli. Probably any organism capable of causing a general infection may be found by blood cultural methods some time or other

in the course of the disease. Very rarely they may be found by direct microscopic examination of the blood. However, cultural methods alone should be depended upon, as they are infinitely more certain and the variety of organism can be readily determined. Clinically, the discovery of bacteria in the blood is of particular value in the early diagnosis of typhoid fever, usually appearing two or three days before the agglutination reaction; in differentiating typhoid and paratyphoid infections and infections simulating them; in diagnosing cryptogenic septic conditions and in the preparation of autogenous vaccines.

Technique for Blood Cultures.—The skin in the bend of the forearm is first prepared as for a major surgical operation. Then the upper arm is compressed by a bandage so that the radial pulse is faintly perceptible. A large glass syringe with a sharp needle at the end, previously sterilized, is then thrust sharply into the most prominent vein, the wall of which can be distinctly felt when the needle is in place. About 20 c.c. of blood are aspirated from the vein, the compress removed, and the needle withdrawn from the vein. The needle is removed from the syringe and about 5 c.c. of blood is then injected into each of four flasks containing bouillon, litmus milk, or other appropriate liquid media, and well shaken. Rigid precautions should be taken to avoid contaminating the culture. The end of the syringe and the top of the flasks should be flamed before placing the blood in the flasks. The flasks are then incubated, examined every twenty-four hours until seventy-two hours have elapsed. If at the end of this time the media are clear the culture is negative. If the fluid becomes turbid before this, subcultures are taken from it and the organism determined by appropriate bacteriological methods (consult Abbott, etc.).

Animal Parasites Found in the Blood.—Many animal parasites have been found in the blood by numerous observers, *e. g.*, trichina embryos, the blood flukes, and the spirochete of syphilis; but for practical diagnostic purposes the important organism sought for to confirm a provisional clinical diagnosis are the malaria plasmodium, the trypanosome of kala-azar, the spirochete of relapsing fever, the trypanosome of sleeping sickness, and the *Filaria sanguinis hominis*.

Malaria.—The best method of hunting for the tertian and quartan organism is by immediate microscopic examination of a fresh specimen of the blood taken just before or during the first part of the chill. If the specimen cannot be examined for several hours, some vaseline may be placed around the edges of the cover-slip to prevent the entrance of air. For better examination or in searching for the estivo-autumnal form, the blood films may be stained by the Romanowsky method. Ross advises extracting the hemoglobin from a large thick drop of blood before staining. Ruge recommends doing this by drying the drop in the air and then placing it in 3 per cent. solution of formalin to which 1 per cent. acetic acid has been added. The hemoglobin is thus extracted and the blood fixed at the same time. It is then washed, stained by the Romanowsky method, dried and restained for thirty seconds with

GENERAL DIAGNOSIS

a solution of borax 5 grams; methylene blue, 2 grams; distilled water 100 c.c., washed, dried, and examined as usual.

The Parasites.—The parasite in its asexual cycle in the blood first appears as small hyaline ameboid bodies in the red cell which frequently assumes a seal-ring like appearance in the estivo-autumnal forms. These bodies become pigmented, the amount of pigment gradually increasing, the ameboid motion becomes slower and the parasite large, occupying most of the cell. The pigmented granules now collect at the centre of the parasite, which commences to have a rosette shape which soon becomes distinctly segmented. When segmentation is complete the spores or segments with a small dark central nucleus may be found irregularly scattered through the red cell, or the segment may burst suddenly through the cell wall as soon as segmentation is complete. During the paroxysm pigment-bearing leukocytes are also frequently observed.

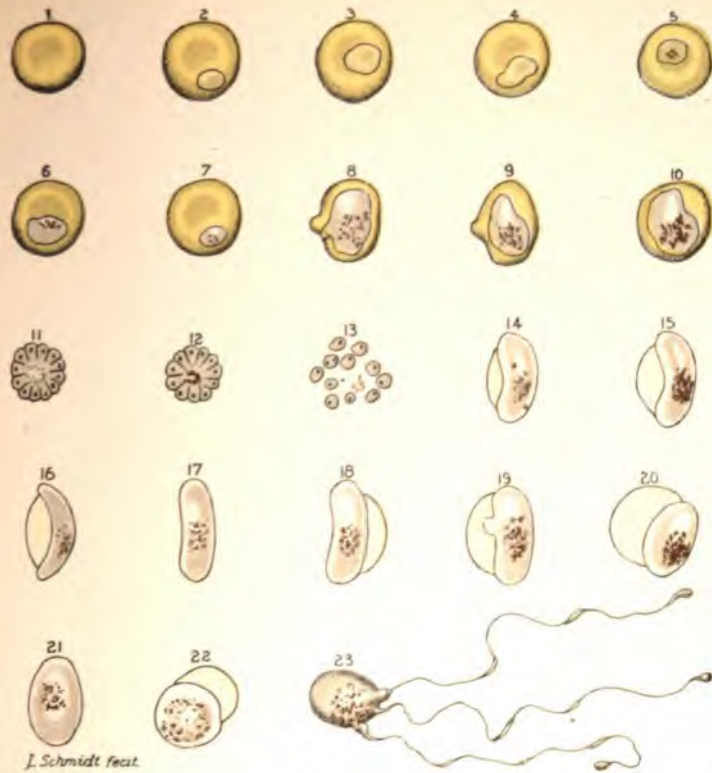
Extracellular Forms.—In the tertian and quartan forms of malaria the intracellular bodies with actively motile pigment granules may appear in the blood free from the erythrocytes without having undergone segmentation. In some of these vacuoles may appear, while others may be broken up into four or five smaller bodies, both an evidence of degeneration. In a few of the organisms, however, flagellation occurs and long actively moving filaments are seen to whip around in the blood stream when examined microscopically. In estivo-autumnal fever crescentic forms appear in the blood stream around some of which is still adherent the delicate outlines of portions of the red cell. These crescents subsequently become rounded or ovoid and some of them still later develop flagella. These flagellated bodies represent the first stages in the sexual cycle of the organism whose later development takes place in the stomach of a mosquito of the genus anopheles.

The several forms of parasite may be distinguished from each other by noting the following characteristics:

	Quartan.	Tertian.	Estivo-autumnal.
1. Cycle	Forty-eight hours	Seventy-two hours	Variable; twenty-four to forty-eight hours
2. Hyaline bodies	Sharply outlined Highly refractive Movement slow	Indistinct Poorly refractive Movement very active	Well outlined. Highly refractive. Movement active.
3. Granules	Coarse Considerable	Fine Many	Fine. Very few.
4. Segments	Six to eight	Fifteen to twenty	Ten to twenty.
5. Red cells	Small Dark	Swollen and pale	Shrunken and brass
6. Extracellular forms	Small	Larger	Crescentic and oval

Trypanosomiasis.—The trypanosomes are frequently the cause of disease in tropical and subtropical countries, affecting both man and animals. Of special interest is the Trypanosoma

PLATE VII
FIG. 1



J. Schmidt fecit.

The Parasite of Estivo-autumnal Fever. (Simon.)

1. normal red corpuscle; 2 to 10, gradual growth of the organism; 11 and 12, segmenting bodies; 13, young forms; 14 to 22, crescents, ovoids, and spherical bodies, with and without bib; 23, flagellated body. Unstained specimen.

FIG. 2



The Parasite of Quartan Fever. (Simon.)

2 to 6, gradual growth of the organism; 7, pigmented body; 8, young form; 9, vacuolated extracellular body; 10, flagellated body.



cause of sleeping sickness. This is seen in the peripheral blood of an affected person as worm-like actively motile unicellular organisms from 10 to 20 μ long. From the anterior end protrudes a long flagellum which in fresh specimens can be seen actively whipping around through the red cells, while from this flagellum a narrow film-like membrane running the length of the body can be distinguished.

The Leishman-Donovan bodies are round, deeply staining organisms found in the large mononuclear leukocytes of patients sick with kala-azar. These bodies probably represent a stage in the development of trypanosomes.

Spirochæta Obermeierei.—These are long, slender, motile organisms, about 40 μ in length, which can be seen in fresh blood smears from patients suffering with relapsing fever.

Filaria Bancrofti.—The adult worms live in the lymphatics of the abdomen and pelvis of man, giving off large numbers of embryos, the *Filaria sanguinis hominis*. In a fresh specimen of the blood these are seen as transparent wriggly but stationary little worm-like bodies among the red cells.

FIG. 114



Filaria alive in the blood. Instantaneous photomicrograph. Four hundred diameters magnification. Four millimeters Zeiss apochromatic. (F. P. Henry.)

The Blood Serum.—The blood serum is examined for the agglutination reactions diagnostic of typhoid, paratyphoid, and Malta fever, and the Wassermann reaction that occurs in syphilis.

The Agglutination Reaction (Gruber-Widal).—The principle of this reaction is based upon the fact that the serum of persons ill with typhoid will cause agglutination or clumping of typhoid bacilli when the proportion of serum to culture is 1 to 10 or more. A dilution of less than this will cause agglutination of the bacilli if the sera of healthy people are employed. Two methods are employed, the microscopic and macroscopic.

Microscopic Method.—To perform this reaction it is necessary to have a virulent fresh bouillon culture of the typhoid bacillus. The cultures can be grown on agar and kept in the ice-box for an indefinite time. When a test is to be performed a subculture is grown in bouillon for eighteen to twenty-four hours at a temperature of 37.5° C.

blood is collected in capillary tubes which can be made by drawing

out either end of a short hollow glass tube and inverting one end about 270° . The blood is collected by capillarity in the bent end of the tube and allowed to stand for several hours or else is immediately centrifuged in order to separate the serum from the cells. The serum is then withdrawn from the unbent end by the capillary pipettes which are also used to make the dilution. These pipettes for diluting the serum are made by drawing out the end of a small caliber hollow tube to a long fine point. Sterile salt solution or bouillon is also necessary. In a porcelain dish with several little hollow cups is placed a drop of the serum to be examined, and to this is added twenty-four drops of the

FIG. 115

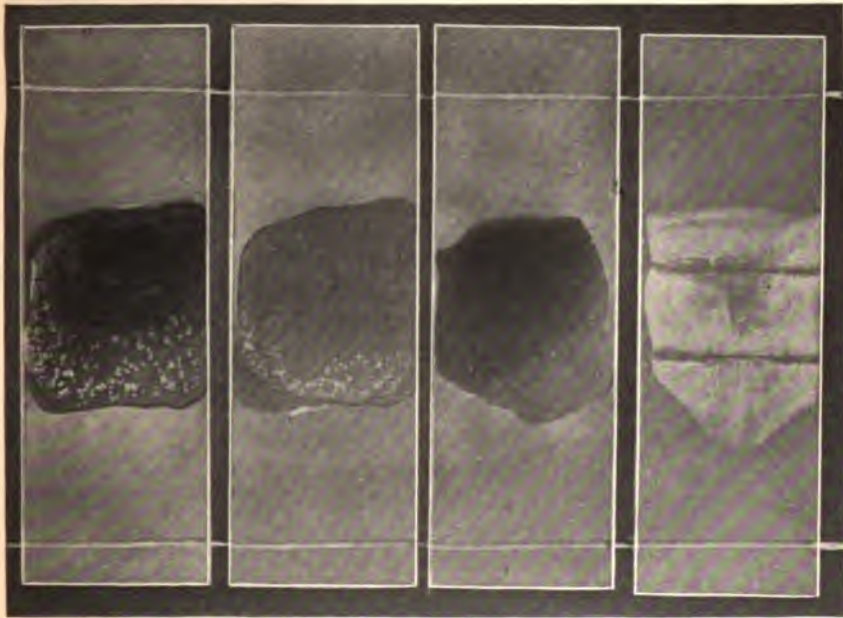


Typhoid agglutination reaction. (Cabot.)

salt solution. Four to ten drops of this are added to another cup, and diluted with an equal number of drops of the salt solution, always using the same capillary pipette both to remove the serum from the collecting tube and to make the dilutions. A platinum loopful of the first mixture is added to a loopful of the bouillon typhoid culture on the centre of a cover-slip. A hanging-drop preparation is made by touching the four corners of a cupped slide with cedar oil, inverting the slide and bringing the four drops of oil in contact with the four corners of the cover-glass so that the hanging drop does not touch the side. A similar procedure is carried out with the second mixture, so that there are two preparations, one a dilution of 1 to 50 the other

1 to 100. These are put aside and examined an hour later. A hanging-drop preparation of the bouillon culture should also be made as a control and examined in an hour.

FIG. 116



Proper blood specimen and reactions in typhoid agglutination test of Bass and Watkins: *A*, a good spread of blood for the test; *B*, negative; *C*, weak positive; *D*, strong positive. (*Progressive Medicine*, March, 1912.)

The Reaction.—In a complete or typical reaction the field when examined with an oil-immersion lens shows the presence of large clumps of motionless bacteria (Fig. 115). No motile organisms can be seen. Clumping and cessation of motion are the essentials of a reaction, provided they occur within a certain time, according to the dilution. When the reaction is feeble or merely suggestive, as in the first stage of typhoid fever, small clumps may appear with a few actively moving bacteria darting across the field or spinning around on their own ends. In this case the test should be repeated in one or two days.

The dried blood may be used as well as the blood serum. A large drop of blood is collected on glazed, non-absorbing paper and allowed to dry in the air. This dry blood can be preserved indefinitely and can also be sent through the mail in an ordinary envelope, so that it affords a convenient method of sending blood to State or municipal laboratories for examination. The reaction is performed as with

Noguchi's Modification.—Noguchi modifies the technique of the Wassermann reaction to increase its accuracy and delicacy by substituting human red cells for those of the sheep, and an antihuman hemolytic amboceptor, prepared from the serum of rabbits who have had repeated intraperitoneal injections of washed human red cells, instead of the hemolytic amboceptor advised by Wassermann. In this way there is obviated the possibility of the hemolytic amboceptors which are found at times in human serum, and which are directed against the foreign sheep corpuscles, having any action hindering the detection of small amounts of syphilitic antibody. Noguchi also recommends as an antigen the employment of lecithin, 0.3 gram dissolved in 50 c.c. of absolute alcohol, to which 50 c.c. of salt solution are added, the filtrate of this solution being used as the antigen. Noguchi further simplifies the technique by using bits of filter paper impregnated with the antigen and the amboceptor instead of the actual solutions.

Only the barest outline of the test is given here in order to show the principles and technique of the reaction in a general way; the preparation of the antigen, complement, and amboceptor requires so much material and the technique is so difficult that only a trained laboratory technician who has had considerable experience, who has the proper facilities, and who is constantly doing the test, should undertake to perform a reaction of such vital importance. Therefore a more elaborate description of the preparation of the solution and of the exact technique is beyond the scope of this book. (See Noguchi.)

Diagnostic Importance of the Wassermann Reaction.—The reaction is positive in from 70 per cent. to 80 per cent. of cases in the primary stage of syphilis, in 95 per cent. to 98 per cent. in the secondary stage, in 70 per cent. to 80 per cent. of latent syphilis, in 90 per cent. of untreated, late, or tertiary syphilis, and in 90 per cent. to 95 per cent. of congenital syphilis. A very large percentage of cases of general paresis and locomotor ataxia give a positive reaction. As a guide to treatment the reaction is also of practical value and is an invaluable procedure before selecting wet-nurses. Occasionally a positive reaction is obtained in normal individuals or in those sick with diseases other than syphilis. It is found frequently in cancer, *e. g.*, in cancer of the stomach 44 per cent. of the reactions are positive, and temporarily in a few cases of scarlet fever.

CHAPTER XXV

THE SALIVA AND SPUTUM

THE SALIVA

THE *saliva* is a white, colorless, odorless, frothy liquid of a specific gravity of from 1002 to 1010. The clinical examination of the saliva is rarely made. The gross amount (normal 1 to 2 liters per day) is sometimes estimated when there is supposedly salivation. The normal alkalinity of the saliva is found to become acid in diabetes, and a few other less important changes may be noted chemically. Potassium sulphocyanide, nitrites, sugar, and ptyalin are occasionally tested for. Microscopic examination of the saliva shows numerous bacteria, schizomycetes, moulds, desquamated epithelial cells, and leukocytes. Occasionally the saliva of normal individuals is examined for the presence of pathogenic organisms which may be the cause of infection transmitted to others, *e. g.*, diphtheria bacilli, or for the presence of abnormal yeasts and fungi in diseases of the mouth.

THE SPUTUM

Sputum is the term applied to all the products of secretion of the respiratory mucous membrane and to other substances that may be brought up through the respiratory tract.

Method of Collection.—Sputum that is to be examined should be collected in perfectly clean vessels which contain no fluid and which can be burnt or readily disinfected. Care should be exercised to prevent the entrance of extraneous substances. It is also necessary to see that the matter sent for examination is derived from the lungs, and is not simply the oral and faucial accumulation. Sputum which upon examination has been found to contain tubercle bacilli should not be allowed to dry in the air, but should be thoroughly mixed with 1 to 20 carbolic acid solution.

General Characteristics of Sputum.—Sputum is alkaline, of a specific gravity varying from 1004 (mucus), 1020 (purulent) to 1037 (serous) and of a consistency varying inversely with the amount of expectoration.

Quantity.—The amount expectorated in twenty-four hours varies from a few c.mm., as in incipient phthisis and in slight bronchial catarrh, to 1000 c.c., as in discharging empyema and in bronchiectasis.

Color.—The color varies with the composition of the sputum and with the nature of the disease. In edema of the lung it is almost trans-

parent and colorless; in acute bronchitis it is white; in pneumonia it is "rusty;" in amebic abscess of the liver it is brownish red or like "anchovy sauce." Red-streaked and prune-juice sputum results from the presence of blood. Red currant-jelly sputum has occurred in malignant disease of the lung and has been found in hysteria. Black sputum is commonly seen in coal miners. Purulent sputum no matter what the cause is yellow or greenish yellow.

Odor.—As a rule sputum has no odor. It is characteristic only in a few cases. That of bronchiectasis, gangrene, and putrid bronchitis is particularly heavy and fetid—a characteristic which renders its origin almost unmistakable.

Varieties and Forms of Sputum.—Sputum may appear in homogeneous or heterogeneous forms.

HOMOGENEOUS FORMS.—Various forms of sputa present a homogeneous appearance.

Mucus Sputum.—This form is usually glairy, clear, and tough. It is seen in the early stages of acute bronchitis and in edema of the lung. A small amount of mucus is expectorated in health, and in cities and smoky towns it is apt to contain black pigment-particles, due to inhaled soot. Mucus sputum is seen in cancer of the lungs.

Purulent Sputum.—This is composed almost entirely of pus. It is seen most typically in a case of empyema discharging through a bronchus. It may also occur in bronchiectasis, in chronic bronchitis, in abscess of the lung, of the liver, or more rarely of the mediastinum when the contents are discharging through a bronchus. It may constitute the discharge of a tuberculous vomica.

Watery or Serous Sputum.—This results from edema of the lung. It is often discharged in large quantities after paracentesis of the chest; "albuminous expectoration."

Sanguineous Sputum.—Blood in the sputum may be seen in greatly varying amounts; it may have many different sources, and it may be of slight or grave significance. It may come from the gums, the nose, the pharynx, or the larynx. There may be cases in which bleeding from the stomach (hematemesis) or esophagus may simulate hemorrhage from the lungs. (See Gastric Hemorrhage and Hemoptysis.)

HETEROGENEOUS FORMS.—These are mixtures of any two or three of the homogeneous forms.

Mucopurulent Sputum.—The most common form of sputum consists of mucus and pus mixed in varying proportions. Such sputa are found in the same conditions that give rise to purulent sputum. *Nummular sputum* is the name given when flat, coin-shaped masses are formed, that sink to the bottom of a vessel containing water, as in phthisis and in chronic bronchitis. When the masses are more spherical, the term *globular sputum* is employed. At times the sputum may be seen to separate into three distinct layers, the upper one frothy, mucopurulent, greenish yellow or dirty green; the middle layer thin and watery; the bottom layer, apparently made up of pus and debris,

opaque, and without air-bubbles. This condition usually points to gangrene of the lung, but it may occur also in bronchiectasis.

Mucoserous Sputum.—A mixture of serum and mucus, as the name implies.

Mucosanguineous, Serosanguineous, and Sanguino-mucopurulent Sputa.—Mucopurulent sputum streaked with blood is frequently seen in phthisis. In pneumonia the rusty sputum is the result of an admixture of mucus and blood pigment, but with changes in the blood coloring matter a yellowish or greenish tinge may be imparted. In certain cases of chronic pneumonia, in which the blood remains longer in the lung tissue, the expectoration has a darker or prune-juice color. The same color may be observed when there is a slight leakage from an aneurism. When pneumonia is accompanied by expectoration of large amounts of blood, it is often of tuberculous origin. Blood may be mixed with the greenish expectoration of gangrene. In chronic valvular disease of the heart, and in oozing from aneurism, frothy mucus containing more or less blood is commonly seen. "Currant-jelly" sputum is more or less characteristic of malignant growths of the lungs, while the expectoration from a liver abscess with amebæ is reddish brown not unlike "anchovy sauce."

Macroscopic Constituents of Sputum.—"Cheesy particles," in size from a millet-seed to that of a pea, are observed in the second and third stages of tuberculosis. They usually contain many tubercle bacilli.

Fibrinous Casts.—These striking, tree-like bodies are found in the sputum of fibrinous bronchitis, and at times in that of pneumonia, phthisis, and of diphtheria when the disease has extended into the bronchi. They are usually mixed with mucus and are rolled into a mass. Their peculiar form is best seen when they are unravelled in water. They are then seen to be a complete mould of a small bronchus with its ramifications. The size varies greatly. They may be many centimeters long. With the microscope leukocytes, blood cells, and alveolar epithelium, and at times Charcot-Leyden crystals and Curschmann's spirals, are found in the meshes.

Spirals.—Under this name are included spiral bodies that are found in the sputum of bronchial asthma, and occasionally in that of pneumonia and of bronchitis and of chronic pulmonary tuberculosis. At the beginning of an asthmatic attack if the sputum is spread on a glass with a dark background they may be seen by the naked eye to have a twisted, spiral form. Microscopically they are found to be made up of spirally arranged mucin in a more or less tight twist, and containing many epithelial cells and eosinophiles. In some of these spirals a clear shining refractive central thread runs through the entire length like a core. The fine fibers comprising the spiral may or may not be closely arranged.

That there is a connection between the spirals and the Charcot-Leyden crystals seems very probable. The latter are absent from the sputum at the beginning of an attack of bronchial asthma; but if a

portion of such sputum is allowed to stand for twenty-four to forty-eight hours, care being taken that evaporation does not take place, crystals will be found. The crystals are often found among the spirals, even when they are seen nowhere else.

Other Substances.—Crystals, elastic tissue, lung tissue, and cartilaginous rings, etc., may be found grossly but should always be examined

FIG. 117

Spirals from bronchial tubes. $\times 80$. (After Leyden.)

microscopically. Concretions from old tuberculous calcareous deposits and foreign bodies are also occasionally seen in the sputum.

Sputum from the Esophagus.—The expectoration in disease of the esophagus is characteristic. It is usually a glairy mucus, often frothy or viscid. It is not coughed up, but after welling into the pharynx is

hawked up. It is abundant in acute and chronic inflammation and in cancer.

Laryngeal Sputum.—The sputum from the larynx is generally scanty. It is colorless, transparent, and not frothy; it is often discharged in small globules; it may be streaked with blood; sometimes pseudo-membranes are coughed up. Laryngeal sputum is found in catarrh and malignant tumors of the larynx.

Microscopic Examination of the Sputum.—**Preparation of the Fresh and Unstained Specimen.**—The fresh and unstained specimen may be prepared for examination with (a) a hand lens, or with (b) a microscope.

Examination with a Hand Lens.—A portion of the sputum is placed upon a piece of window glass, about 15 cm. square, which has been painted black on its under surface or is laid upon a black ground. A smaller piece of window glass, about 10 cm. square, is placed over the large glass so as to press out the sputum in a thin layer. The thin layer is then examined with a hand lens or with the unaided eye, for foreign particles, elastic fibers, animal parasites, etc.

FIG. 118



Ordinary cover-slip forceps.

Fresh Examination with the Microscope.—A particle or a drop of the sputum is placed upon a glass slide and covered with a cover-glass, by means of which it is pressed until flattened into a thin layer. The specimen is then examined first with the low power of the microscope and afterward with the high power.

Preparation of the Dried Specimen.—A small amount of the purulent portion of the sputum is spread in a thin and uniform layer on a perfectly clean cover-glass or slide by means of forceps, needles, a platinum loop, or other instruments, which previously must be sterilized by being held a moment in the flame of a Bunsen burner or spirit lamp. The sputum is then dried in the air, or more quickly by holding the cover-glass some distance above the flame of a burner or lamp. Finally it is passed three or four times through the flame, and so "fixed."

Staining of the Sputum.—Methylene blue is the stain most generally employed. When the leukocytes are to be studied, a stain of eosin and hematoxylin or Romanowsky's stain may be employed, as if staining a blood film. For the detection of tubercle bacilli the specimen is stained with special stains.

Microscopic Constituents of the Sputum (Fig. 120).—*White Blood Corpuscles.*—These are present in all sputum, but in varying numbers and size. They are usually of the polymorphonuclear variety,

and are most abundant in purulent sputum. Often they contain fat drops and pigment particles. Frequently many of the leukocytes contain large numbers of organisms, *i. e.*, pneumococci, influenza bacilli, or tubercle bacilli.

FIG. 119

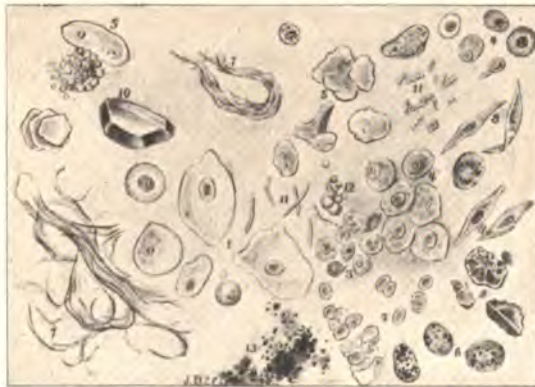


Boston's slide holder.

EOSINOPHILE CELLS.—These are frequently found in large numbers in the sputum during the exacerbations of bronchial asthma. They are also present in the sputum in acute and chronic bronchitis and in phthisis.

RED-BLOOD CORPUSCLES.—These are to be found in most sputa, but they may be so few as not to give a red color. The source is often high up in the respiratory tract. When they are present in large

FIG. 120



Various objects from sputum: 1, squamous epithelium; 2, red-blood corpuscles; 3, polynuclear leukocytes; 4, alveolar cells; 5, myelin cells; 6, heart-failure cells; 7, elastic tissue fibers; 8, cylindrical epithelium; 9, hematoidin crystals; 10, phosphate crystals; 11, fungi; 12, fat globules; 13, free pigment. (Original observation.)

numbers they indicate always some pulmonary disease. Usually each cell is well preserved, but they may appear as pale bodies or as rings, the pigment remaining in the sputum as pigment particles, as in pneumonia.

EPITHELIUM.—Two general varieties of epithelium are found in the sputum—squamous and cylindrical. The former comes from the mucous membrane of the mouth, pharynx, and larynx.

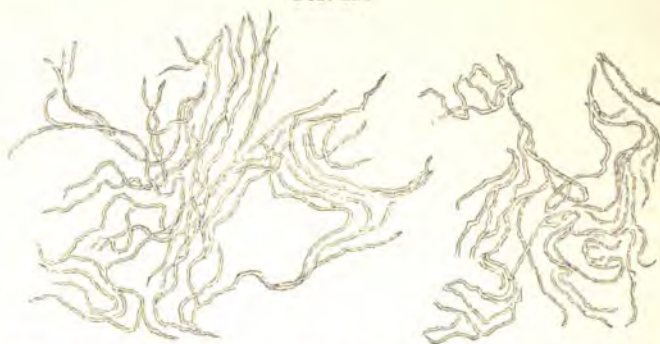
Cylindrical cells are found in inflammations of the trachea and bronchi, or of the posterior nasal fossa, a locality where ciliated epithelium exists normally.

"*Alveolar*" epithelium, containing numerous pigment, fatty, or myelin granules, not only occurs in almost all pulmonary affections, but also at times in normal sputum. They are round or oval cells, somewhat larger than a leukocyte, with one or two oval nuclei.

Frequently in cases of heart disease with failing compensation the alveolar cells may contain large amounts of hematoidin granules, (heart-failure cells) (see Fig. 120).

ELASTIC FIBERS.—The presence of elastic fibers in sputum is of much import, denoting destruction of the lung tissue. In the great majority of cases their presence is the result of tuberculosis, but they are also seen in cases of abscess of the lung, bronchiectasis, and occasionally in

FIG. 121



Elastic fibers of lung tissue obtained from sputa after digestion in caustic soda.
(Drawn by Dr. John Wilson.)

pneumonia and gangrene of the lung. They are sometimes mistaken for fat crystals. They are to be recognized by the double contour and the curling ends, but best by their alveolar arrangement, as it is then positive that they have not come from extraneous sources.

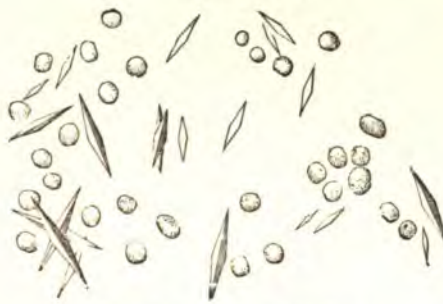
Detection of Elastic Fibers.—A small amount of thick, purulent portions of sputum is pressed out in a thin layer between two pieces of plain window glass. The particles of elastic tissue appear on a black background as grayish-yellow spots, and can be examined *in situ* under a low power. Or the upper piece of glass may be slid off until the piece of tissue is uncovered, when it is picked out and examined on a microscope slide, first with a low power and then with a higher power. This method is much easier of accomplishment and is quite as satisfactory in results as the one generally employed—boiling an equal quantity of sputum and solution of caustic potash (8 to 10 per cent.) for a short time, and then allowing the mixture to stand for twenty-four hours in a conical glass. The elastic tissue remains intact and is found in the sediment.

CONNECTIVE TISSUE AND CARTILAGE.—In fragmentary bits these are rare constituents of sputum. The former may occur with abscess or gangrene of the lung, and the latter when there is ulceration of the larynx, trachea, and bronchioles.

CRYSTALS.—*Charcot-Leyden Crystals.*—These are octahedral, sharply pointed colorless or slightly bluish crystals, soluble in warm water, alkalies, acetic acid, and mineral acids. They appear grossly as small yellowish bodies, not unlike grains of sand; under the microscope they are unmistakable. They occur most abundantly during and after an attack of bronchial asthma, but may also be seen occasionally in the sputum of acute and chronic bronchitis and tuberculosis.

Cholesterin Crystals.—These crystals are similar to those of cholesterin found elsewhere. They may be present in the sputum of tuberculosis, simple and hydatid abscess of the lung, and in pus from an old abscess of a contiguous organ which has entered the lung.

FIG. 122



Charcot crystals. (Scheube.)

Hematoidin Crystals.—These have a brownish-yellow or ruby-red color, and are either in the form of small rhomboid prisms or of fine needles arranged singly or in bunches of various shapes, or they may occur as free pigment particles without crystalline form; smaller particles may be contained within a leukocyte. Their presence indicates that blood has remained in the respiratory tract for some time before being expectorated, or that an abscess has discharged into a bronchus.

Fatty Crystals.—These appear as long, thin needles, and occur either singly or in bundles. They are found in pus, as in gangrene, putrid bronchitis, bronchiectasis, and tuberculosis. They dissolve in ether and in boiling alcohol.

Tyrosin Crystals.—These have been found in the sputum of putrid bronchitis and of an empyema discharging into the lung, and usually occur in conjunction with leucin. (See Urine.)

Oxalate of Lime and Triple Phosphates.—These have been noted occasionally in sputum; the former in cases of diabetes and of asthma, the latter in alkaline sputum.

ANIMAL PARASITES.—*Echinococcus cysts*, generally broken into fragments and only very rarely in a perfect whole, are to be found in the sputum when there is rupture of a cyst of the liver or of the lung into a bronchus. Scolices and free hooklets may be recognized, and pieces of the cyst wall will be known by their remarkable formation. Their presence is of great clinical value.

Distoma hematobium eggs may occur in sputum.

Distoma westermanii or *pulmonale* are found in the sputum of Japanese suffering from this infection, which much resembles phthisis. Both the worm and the ova may be present in the sputum.

Entamoeba Dysenteriae.—A full description of amebas will be given in the chapter on Dysentery. The amebas are the same in every respect when found in the sputum. Upon exposure to the air the sputum becomes thin, syrupy, and oily, looking much like anchovy sauce. It is alkaline and of a sweetish odor, never putrid. Microscopically there will be found, besides the amebas, red-blood corpuscles, leukocytes, alveolar and oval epithelium, and bodies looking like degenerated liver cells without a nucleus; occasionally elastic fibers, hematin, leucin, tyrosin, and Charcot-Leyden crystals and bacteria are seen. The sputum should be examined as soon after discharge as possible, and in the interim should be kept at a temperature of 30° to 35° C. If examined on a warm stage, active movements of the amebas will be kept up much longer.

VEGETABLE ORGANISMS.—Certain moulds, *e. g.*, *mucor* and *aspergillus*, have been considered to cause disease of the lungs, but nothing very definite has been discovered except that they are associated with cavity formation. *Leptothrix*, occasionally found in sputum, is probably taken up from the mouth. *Oidium albicans* may be a constituent of the sputum, but usually it comes from the saliva.

Sarcinae Pulmonales.—*Sarcinae pulmonales* may be seen in sputum. They are larger than *sarcinae ventriculi*, with which they have no connection. They have no pathological significance.

Streptothrices.—*Streptothrix actinomyctica*, or ray fungus, is found in the sputum of infected patients. The characteristics of the sulphur granules can be demonstrated readily by crushing them under a coverslip. This is usually sufficient for a diagnosis, but if the morphology is to be more carefully studied the fungus may be stained by Gram's method for three minutes after having been first immersed in aniline-gentian water for ten minutes and later decolorizing the rest of the smear by repeated washing in xylol-aniline (1 to 2) until clear. The mycelium is then a blackish-purple or violet color. Numerous other streptothrices have been found in the sputum of cases presenting the clinical signs of tuberculosis, chronic bronchitis, or pulmonary gangrene.

Bacteria.—Numerous forms of pathogenic and non-pathogenic bacteria are found in the sputum and saliva of healthy individuals. Hence the diagnostic significance of pathogenic bacteria in the sputum

is but slight. Furthermore, most of the pathogenic bacteria can only be satisfactorily demonstrated by cultural methods, so that their clinical diagnostic import is still lessened.

The *Diplococcus pneumoniae*, a paired, lancet-shaped coccus with a distinct enveloping capsule, though occurring normally in the saliva and sputum of a great number of people, is usually found in enormous numbers in the sputum of cases of lobar pneumonia. At times the discovery of these cocci in large numbers in the smears of the sputum confirms the provisional diagnosis of an obscure case.

FIG. 123



Actinomyces.

Tubercle Bacillus.—The great exception to the above statement, however, is found in extreme diagnostic importance of tubercle bacilli in the sputum. Very few cases of pulmonary tuberculosis fail to show tubercle bacilli in the sputum if repeated examinations are made. The failure to find them after repeated searching does not necessarily prove that tuberculosis is absent, but the discovery of them is practically pathognomonic, as the smegma bacillus and the lepra bacillus, the only two organisms, both acid resisting, with which they may be confounded, are so infrequently found in the sputum under ordinary circumstances in this country. The smegma bacillus only is at all likely to cause difficulty so that the differentiation between it and the tubercle bacilli may sometimes require animal inoculations or cultural methods.

The number of bacilli in the sputum varies exceedingly without any relationship to the severity of the local process, so that little definite

knowledge is gained from the number in a smear or a field, nor from the study of their morphology. The organism is rod-shaped, straight, or slightly curved, without motion, varying in length from 2μ to 5μ . It often has a beaded appearance when stained. Bacilli presenting this appearance are supposed by some to be undergoing degeneration. Branching forms of the tubercle bacillus are seen at times. The bacillus of tuberculosis cannot be recognized in the sputum unless stained. It is recognized by the fact that when once stained, it is not decolorized by acids.

Staining the Tubercle Bacillus.—The sputum is spread out on a glass plate with a black background and several small cheesy masses, or if they are absent, several portions of the sputum from different parts of the spread-out material are picked up on a platinum wire and spread lightly on the slide or cover-slip. The specimen is then dried, and fixed by passing it through the flame three times.

I. Ziehl-Neelsen Method:

Solutions used:

- | | |
|---|----------|
| A. Carbol-fuchsin solution. | |
| Distilled water | 100 c.c. |
| Carbolic acid (crystalline) | 5 gm. |
| Alcohol | 10 c.c. |
| Fuchsin in substance | 1 gm. |
| B. Decolorizing solution of nitric acid 25 per cent. in 70 per cent. alcohol. | |
| C. Contrast stain of 1 per cent. watery solution methylene blue. | |
- The tubercle bacilli are stained red, the other bodies blue.

Cover the dried sputum with the desired stain, and steam gently for two minutes over a low flame. Pour off the excess of stain, then cover the stained sputum with the decolorizing agent, wash and if the smear still retains its color, decolorize and wash a second time. Repeat until there is no further color. Counter-stain for thirty seconds. Wash with water and cover with a clean cover-glass. Examine the smear with the oil-immersion lens, and preferably with the movable stage.

II. Gabbet's Method:

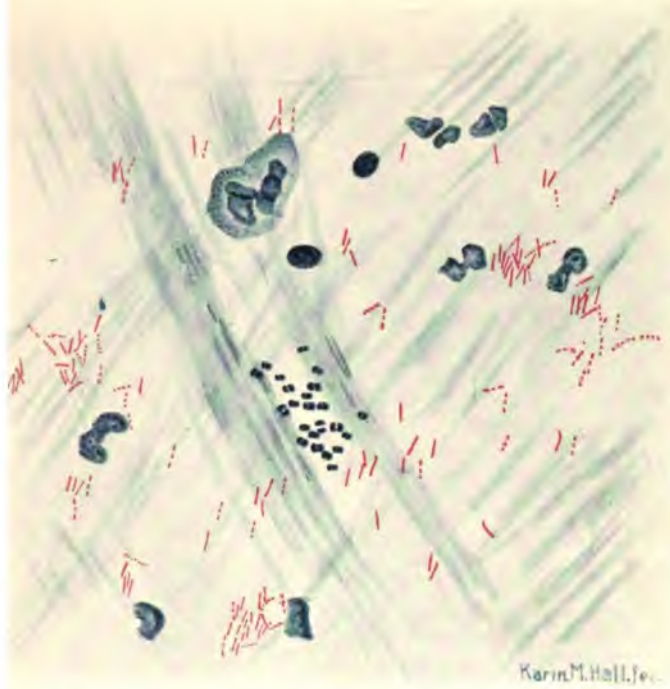
Solutions used:

- | | |
|--------------------------------------|---------|
| A. Carbol-fuchsin solution as above. | |
| B. Methylene-blue solution. | |
| Methylene blue | 5 gm. |
| Sulphuric acid | 25 gm. |
| Distilled water | 75 c.c. |
- This solution is apt to decompose with age.

The cover-glass is prepared and stained with the carbol-fuchsin solution, which is poured off as above. Then (instead of decolorizing with nitric acid) the slip is washed for twenty to thirty seconds in the methylene-blue solution until a faint blue replaces the red tinge in the (slip) sputum; the excess of the solution is washed off in water, and the slip is mounted and examined as above. The tubercle bacilli are stained red and the other bodies blue. Decolorization with absolute alcohol, in addition, must be employed to eliminate the presence of other acid-resisting bacilli.

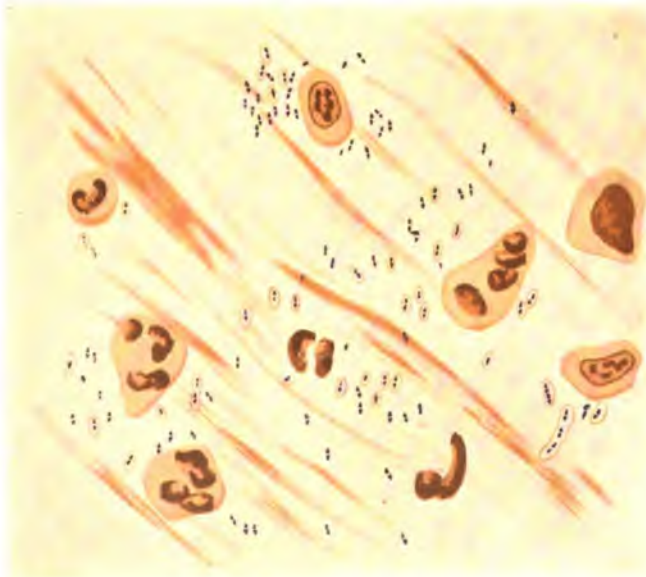
PLATE VIII

FIG. 1



erle Bacillus (Red). Diplococci (Unclassified) Blue.

FIG. 2



III. Pappenheim's Method:

A. Carbol-fuchsin solution as above.	
B. Corallin (rosolic acid)	1 gm.
Absolute alcohol	100 c.c.
Add methylene blue to saturation.	
Glycerin	20 c.c.

Stain with the carbol-fuchsin as above, pour off the excess of stain, and add the corallin solution. Fresh solution is added several times until the red tinge has disappeared from the spread. Tubercle bacilli are stained red; smegma bacilli blue. This method differentiates the two bacilli absolutely in the great majority of cases, a factor which Gabbet's method lacks.

Antiformin.—If but few bacilli are present they may be concentrated by dissolving out the mucoid material and bacteria other than the acid-fast bacilli, in a solution of antiformin, a 10 per cent. solution of sodium hypochlorite, containing 10 per cent. of sodium hydrate. An equal amount of this solution is added to the sputum and either gently warmed until the mucus has disappeared, or left on overnight. The remaining material is then centrifuged, the excess of antiformin poured off from the sediment and water added. The supernatant fluid is poured off several times and fresh water added centrifuging after each addition of water. The sediment is then placed on a cover-slip, dried, fixed, and stained as outlined above. If the sediment does not stick to the slip, it probably has not been washed sufficiently. A thin coating of egg albumen may also be placed on the slip to better hold the sediment. The antiformin does not kill the bacilli, so care should be taken to prevent the escape or the splattering of the fluid over the centrifuging tubes and in the air. The sediment may be used for animal inoculation. The concentration of the bacilli results in practically all the tubercle organisms in the sputum remaining in the sediment, so that the chances of missing them is reduced to a minimum, and furthermore, all the sputum is examined, not merely a few portions of it picked up at random.

Chemical Examination of the Sputum.—The chemical examination of the sputum has proved of but little clinical value. Recently the presence in the sputum of albumin has been shown to be indicative of an inflammatory process somewhere in the respiratory tract, and is supposedly an important diagnostic sign of pulmonary tuberculosis in the absence of any other demonstrable inflammatory process. The mouth should be well cleansed to exclude extraneous matter, and mucus from the nose and throat. The sputum collected should only be brought up by coughing and should contain as little saliva as possible. To demonstrate the albumin, shake the sputum with a 3 per cent. solution of acetic acid until the mucus has disappeared. Filter and wash the filtrate with 3 per cent. acetic acid. Test the filtrate for albumin with potassium ferrocyanide, or add sodium hydroxide until only faintly acid and test by boiling. The greater the precipitation upon boiling the greater the inflammatory process which causes the sputum.

CHAPTER XXVI

THE STOMACH CONTENTS

THE chief functions of the stomach, secretory and motor in nature, are studied largely by the examination of the stomach contents removed by the stomach-tube.

Mode of Procedure.—A test breakfast (Ewald) or a test dinner (Leube) is administered, or the *fasting* stomach contents are removed. *Ewald's test breakfast* consists of one or two slices (35 gms.) of bread without butter and a cup of weak tea ($\frac{1}{3}$ liter) without sugar or preferably the same amount of water. The *Leube-Riegel test dinner* includes a large plate of soup (400 c.c.), a large portion of beefsteak or other meat (150 gms.), some potatoes (50 gms.), and a roll. (2) Remove the contents of the stomach in from forty minutes to one hour after the breakfast, and three or four hours after the dinner, by aspiration or by expression.

Aspiration consists in the withdrawal of the stomach contents by suction: either with the ordinary stomach-pump, by means of a bottle exhausted of air as employed for paracentesis, and connected with the stomach-pump, or by connecting the sound with a hand-ball aspirator or Politzer bulb, or by the suction that can be secured by compression of the bulb of the stomach-tube and the pinching the distal end of the tube. *Expression* consists in compressing the abdominal muscles, imitating the act of straining in defecation. The patient takes a deep inspiration and then contracts the muscles as above. If the tube is sufficiently long, it can be bent, so as to assist expression with siphonage. Aspiration is less disagreeable to the patient, and is necessary when the stomach contents are not sufficiently fluid to flow easily. A soft-rubber tube with two good-sized openings near its distal extremity should be selected. Stockton suggests a tracing of rings around the tube one inch apart, beginning 20 inches from and ending 30 inches from the lower extremity, for the purpose of measuring the length of tube inserted. In healthy adults the distance from the incisor teeth to the lower border of the stomach is about 22 inches; in dilatation it may be from 24 to 30. The distance is partly determined by success in the siphonage. If the return flow of fluid does not take place, it is well either to withdraw the tube or push it farther on; for if too long it may curve above the level of the fluid, or if too short it may not reach the fluid.

It is sufficient simply to moisten the tube, since the saliva acts as a lubricant. The patient should be seated, and the tube at once passed

to the back of the pharynx, and pushed toward the esophagus or lower pharynx, and if the patient is instructed to swallow and to breathe slowly, it is rapidly carried downward by deglutition. Mucus that accumulates in the mouth after the tube has been passed should be allowed to dribble outward and not be swallowed. The head should be bent forward. After the tube has descended below the level of the fluid in the stomach the contents are removed by aspiration or expression into a convenient vessel.

FIG. 124



Illustrating expression and lavage.

Physical and Chemical Examination of the Stomach Contents.—
A. Physical Examination.—**QUANTITY.**—The quantity of fluid, after digestion of a test breakfast (given after a twelve-hour fast) has continued for one hour, should be from 30 to 40 c.c. If over 80 c.c. then

there is present either hypersecretion, motor insufficiency, or pyloric obstruction. The filtrate is clear and yellow or yellowish brown in color. If digestion is normal, the fluid should contain free hydrochloric acid, pepsin, and rennin (the milk-curdling ferment), but no lactic acid. Albuminoids should be converted into proteoses and peptone, and starches into achroödextrin, dextrose, or maltose, though small amounts of erythrodextrin are usually present.

REACTION.—The normal reaction of the contents of the stomach is acid; it may be alkaline in cases of hemorrhage or in the vomiting known as water-brash.

ODOR.—The odor is sour normally; it may be aromatic from the presence of the fatty acids; fecal, in obstruction of the bowels with fecal vomiting; and finally, may indicate the nature of poisonous ingesta—ammonia, phosphorus, carbolic acid. The dark, frothy material from a dilated stomach is of a foul yeasty or putrid odor.

INSPECTION OF THE STOMACH CONTENTS.—By ordinary inspection the *quantity* and the *character* of the vomitus or stomach contents are noted. The most important evidence of secretory change is achylia gastrica (absence of secretion), in which the bread is returned in the form in which it was taken, except it appears water-soaked.

An abnormally great quantity of solid matter and a small amount of chyme indicate abnormal retention, which is usually due to motor weakness or to pyloric obstruction. Sometimes when there is a large residue in the stomach, the contents separate into three layers: the uppermost consisting of mucus or undigested food; the second, generally the thickest, of fluid; and the lowest of chyme. Such a formation points to abnormally long retention as the result of stenosis and consecutive dilatation, or to motor weakness. A chemical examination of the stomach contents is necessary to satisfactorily study the gastric secretions. Undigested food should not be present six hours after an ordinary meal.

We can often discover by inspection whether food is brought up by *vomiting* or by *regurgitation*. When digestion is normal and the individual has eaten meat, regurgitation of food from the esophagus can be differentiated from vomiting by the appearance of muscle fibers; if the food is vomited the fibers are in a state of disintegration; if not, they are intact.

Mucus.—Mucus is found in small quantity normally, but is increased in catarrhal affections of the mouth, throat, or stomach. When its source is the mouth, *saliva* also is generally present. Mucus is recognized by its stringy, tenacious character, when a clean glass rod is stirred in the gastric contents.

Bile and intestinal juice may be regurgitated into the stomach as the result of violent emesis, or when the pylorus is much relaxed, or in stenosis of the duodenum below the common duct, when bile is present in large quantities if the stomach is dilated. Bile is grossly

recognized by its greenish-yellow color. Persistent absence of bile in the vomitus is an indication of pyloric stenosis.

Blood.—Blood is found in ulcer, cancer, acute, especially toxic, gastritis, injuries to the mucous membrane from the use of the tube for expression, and after violent retching. It is also common in cirrhosis of the liver, and may occur in purpura, the hemorrhagic diathesis, and in yellow fever. Blood mixed with gastric mucus may come from the lung, the act of coughing having excited vomiting.

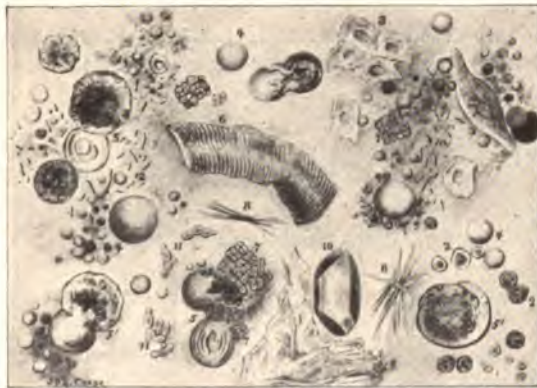
Gross blood may stain the gastric contents bright red, or it may be partially digested and intimately mixed with the stomach material, giving rise to a "tarry" or "coffee-ground" appearance. Small amounts of unaltered blood may be recognized by microscopic examination or by chemical test if in very small quantities (see *Feces*), or if largely digested.

Pus.—Pus is rarely present in sufficient quantity to be detected by the naked eye, but it sometimes occurs in phlegmonous gastritis and when an abscess has ruptured into the stomach or mouth.

Fecal matter is vomited in complete obstruction of the bowels. It is recognized partly by its appearance and partly by its odor.

Worms are sometimes vomited; the round worms not so very infrequently; oxyurides and ankylostomata rarely.

FIG. 125



Microscopic appearance of stomach contents: 1, red-blood corpuscles; 2, leukocytes; 3, squamous epithelium; 4, fat globules; 5, starch granules; 5', starch changed by action of the gastric juice; 6, muscular fiber; 7, *Sarcinae ventriculi*; 8, fat crystals; 9, pieces of orange; 10, phosphatic crystal; 11, yeast fungi; 12, bacilli and micrococci.

MICROSCOPIC EXAMINATION.—The illustration (Fig. 125) shows the various matters that may be found in vomited matter. Briefly, they are columnar and squamous epithelium, white-blood corpuscles, acted on by gastric juice, and red-blood corpuscles. The corpuscles

are usually isolated. The red are rarely perfect, and in the white little more than the nucleus remains. The pus cells may come from septic conditions of the mouth or lungs, but usually indicate ulcerative conditions of the stomach, notably ulcerating cancer. From the food we may also find muscle fiber, fatty globules and fat needles, elastic and connective-tissue fibers, starch granules, and vegetable cells. Muscle fibers are recognized by their transverse striation. Fat globules are soluble in ether, and are recognized by their refracting power. Starch granules stain blue with iodopotassic iodide solution. Crystals of leucin, tyrosin, and cholesterol are frequently seen in vomitus.

In addition, fungi of many forms are found, as the mould fungi, the yeasts, and fission-fungi. The latter are recognized after staining by the iodopotassic iodide solution, which colors them blue. The most important fission-fungi are *Sarcina ventriculi*. They are of dark gray tint, stain malugany brown to reddish brown with the above-mentioned solution, and resemble in shape corded bales of goods. Yeast and *sarcina* are present when fermentation is in progress, and hence indicate dilated digestion from motor insufficiency. *Sarcinae* are usually also an indication of dilatation of the stomach from a benign cause, since they flourish only in stagnating contents which are decidedly acid, and these conditions are afforded by benign pyloric stenosis, but not by carcinoma.

The *Oppler-Boas bacilli* have a contrary significance. These are long, thick, club-like organisms, often pointed at one end, and usually showing a decided Brownian movement. They are usually found in cancer cases, and often in enormous numbers, and are of about the same value in diagnosis as the presence of large amounts of lactic acid.

Fragments of tumors, giving conclusive evidence of malignant disease, are at times found in vomitus and gastric contents.

B. Chemical Examination.—A chemical examination is made to determine (1) the presence of free acids; (2) the degree of total acidity of the stomach contents; (3) the presence of free HCl; (4) the presence of lactic acid; (5) the presence of volatile acids; (6) the presence of pepsin; (7) the presence of rennin; (8) the character of the carbohydrates.

Hydrochloric acid is the normal acid of the gastric juice. Normally lactic acid is found during the first half-hour of digestion when starches have been taken; but when only meats have been taken, lactic acid is not found so early.

1. *Free Acids.*—The most sensitive test for free acids is *Congo red*. Filter paper soaked in a saturated solution of the dye and allowed to dry is turned a deep blue if free acid is present. Lactic, butyric, or acetic acid—organic acids—one or all, may be present without HCl. HCl and one or more of the organic acids may be present together.

Free acidity may be due to an inorganic acid (hydrochloric) or organic acids (lactic, butyric, acetic).

2. *The Total Acidity.*—This is determined by titration. The stomach contents must be filtered. Fill a Mohr burette with a decinormal solution of caustic soda.¹ To 10 c.c. of the filtrated gastric fluid add 2 drops of a 1 per cent. alcoholic solution of *phenolphthalein*.

Allow the caustic soda solution to drop slowly from the burette into the fluid until permanent rose-red color is produced and does not disappear on shaking. 4 to 6 c.c. of the caustic soda solution are required to neutralize the acid at the height of *normal digestion*. The degree of acidity is expressed in terms of the number of c.c. of decinormal sodium hydrate solution necessary to neutralize the acid in 100 c.c. of gastric contents. Thus if 4.3 c.c. neutralize 10 c.c. the total acidity will amount to 43, or if 6 c.c. are required to 60. If more or less than the amount just indicated of the alkaline solution is required to neutralize the acid the total acidity is increased or diminished and hence is abnormal.

3. *Free Hydrochloric Acid.*—If only a small quantity of gastric contents are obtainable the free HCl may first be estimated and then the total acid, using the same filtrate. The phenolphthalein is added after the titration for the free HCl, but the total acid will represent the entire amount of decinormal solution that is used. 10 c.c. of filtered gastric contents should be used in making estimations of the acidity of the stomach contents, but if not obtainable 5 c.c. may be employed, multiplying all results by two when this amount is used. If there is insufficient quantity to filter, the unfiltered contents may be used.

Topfer's test for the detection of free HCl is performed as follows: dimethylaminoazobenzol is employed in a 0.5 per cent. solution of alcohol. To a few c.c. of filtered stomach contents 1 to 4 drops of the reagent are added in a test-tube or beaker. If hydrochloric acid is free a rose-red color is produced when the filtrate is added to the reagent. The drug reacts to HCl only when the latter is in a free state. If organic acids are present in a concentration of from 0.5 to 0.8 per cent. a distinct reaction may be brought about, and smaller amounts give a decided orange color.

Phloroglucin-vanillin, introduced by Gunzburg, is a very sensitive test for HCl. It does not react to organic acids, and is now generally relied upon. As the Gunzburg reagent does not keep well, it is best put up as follows: phloroglucin, 2 gm.; alcohol, 30 c.c.; vanillin, 1 gm.; alcohol, 30 c.c. These are mixed in equal quantities as needed. One drop of the reagent is put into a porcelain dish with two or three drops of the stomach filtrate. Upon *cautious* heating over a *small* flame a beautiful carmine colored surface is formed, especially at the edges

¹ Decinormal solution of sodium hydrate is of the strength of 4 grams of pure sodium hydrate to the liter of distilled water. Since sodium hydrate readily absorbs water the solution should always be made of approximate strength and then corrected by titrating against a decinormal oxalic acid solution (6.285 grams oxalic acid to a liter of distilled water).

solution that has been employed to obtain this reaction, multiplied by 10, is subtracted from the total acidity to secure the amount of combined acid present in the filtrate. To estimate the acid salts the amount of free HCl is subtracted from the combined acids. For example, the results may be put down as follows:

Total acid	60
Alizarin acidity	30
	—
Therefore combined acids	30
Free HCl	26
	—
Therefore acid salts	4

4. *Lactic Acid*.—If the stomach contents are colorless, apply the following tests; if they are yellowish, make an ethereal extract, as described below, and then apply the tests. The presence of lactic acid may be determined by Uffelmann's test: Mix 1 drop of pure carbolic acid with 5 drops of a diluted solution of neutral ferric chloride; add sufficient water to render the whole of an amethyst-blue color; to this add a few drops of the gastric fluid; a mere trace of lactic acid will change the blue to a light (canary) yellow or a greenish yellow. The test for lactic acid is obscured by phosphates and simulated by excess of HCl or when glucose or alcohol is present in the gastric juice, therefore the lactic acid should be removed by extracting with ether, as follows: to 10 c.c. of gastric contents add 50 c.c. of ether; shake well for ten minutes. Evaporate on a water-bath, dilute with distilled water up to the amount of filtrate employed (10 c.c.) and add the reagent. Kelling's test is more delicate: add 1 drop of a solution of 5 per cent. solution of ferric chloride to 50 c.c. of water and 5 c.c. of filtrate; the presence of lactic acid causes a greenish coloration.

5. *The Volatile Acids*.—These acids are best detected by their odor, their volatility, and their reaction.

Butyric acid is recognized by the pungent odor of rancid butter given off when the stomach contents are evaporated. It is recognized by the following reaction: to a small quantity of the evaporated residue of the ethereal extract add a small quantity of alcohol and 2 drops of strong sulphuric acid; heat for a short time; a characteristic smell of butyric ether, like that of "pineapple rum," is given off.

Acetic acid is recognized by its odor, particularly after heating the solution. It may be detected as follows: an ethereal extract of the gastric contents is dissolved in water; after the watery solution has been neutralized with a dilute solution of sodium hydrate, neutral ferric chloride solution is added, when a blood-red color results if acetic acid is present.

6. *Pepsin*.—If HCl is present, add 25 c.c. of a gastric filtrate to 0.05 gm. of egg albumen. Allow digestion to take place for three hours at 37° to 40° C., at the end of which time it should be digested. If HCl is absent, pepsinogen, the zymogen of pepsin, is found alone. Add 2 drops of a 25 per cent. HCl solution to 25 c.c. of the gastric contents.

Add to this solution a small portion of egg albumen. If it is dissolved, pepsinogen was converted into pepsin by HCl.

7. *Chymosin (Rennin) (the milk-curdling ferment).*—This may be detected as follows: 5 to 10 c.c. of cows' milk are added to 3 to 5 drops of filtrated gastric juice; the mixture is placed on a water-bath and heated to 37° or 40° C. If the ferment is present, the casein of the milk is precipitated in flakes or in a curdy mass in from twenty to thirty minutes.

Chymosinogen.—If the enzyme is absent the zymogen is tested for by treating the milk with 10 c.c. of neutral filtered gastric contents and then adding 2 or 3 c.c. of a 1 per cent. solution of calcium chloride. This is heated as above and if the xymogen is present a thick cake of casein will occur.

8. *Lipase.*—Normally present in the stomach in small amounts the difficulty of testing for lipase makes such a procedure of much less clinical value than the tests for acids, pepsin, etc.

9. *The Carbohydrates.*—Add a few drops of Lugol's solution to the gastric contents; if starch is present, it turns blue; if erythrodestrin, brown. If the digestion has proceeded so far as to change starch into achroödextrin maltose, the iodine hue remains unchanged. Maltose and dextrose are tested for with Fehling's solution. The digestion of starches varies inversely with the amount of HCl present; in an acidity they are completely digested an hour after a test breakfast; in hyper-acidity there may be little digestion.

The Motor Power.—When digestion is normal, the stomach contents removed six hours after a Reigel test dinner are neutral and contain only a few flakes of mucus. At the end of five hours the stomach contents are acid and contain some undigested muscle fibers and starch granules. If the stomach contains undigested food at the end of six hours, and the contents are acid, a delay in digestion is indicated.

The study of the motor power by means of test meals is unquestionably the most practical and the most exact method as yet devised. A lack of motor power is indicated by (1) the presence of food in the fasting stomach before breakfast; (2) the presence of food in the stomach two hours after a test breakfast has been taken; (3) the presence of food in the stomach six hours after a test meal. The degree of retention can be readily determined by the removal of a test dinner, six, seven, eight, or more hours after ingestion. In order to make the presence of food remnants more readily visible, some food which is not readily digested can be added to the Reigel test dinner, as raisins, prunes, or currants. Such articles of food may be taken alone without the test meal, and are readily recovered later by gastric lavage.

Donald and Sievers have suggested also the ingestion of 15 grams of sodium which should appear as sodiumuric acid in the urine in from forty-five to seventy-five minutes. The urine, treated with a few drops of ferric chloride, turns a violet color in the presence of the acid. In the reaction shows motor insufficiency.

The Resorptive Power.—Penzoldt and Faber recommend the administration of 0.2 gm. of chemically pure potassium iodide a short time before dinner. The saliva acidified with nitric acid is tested for iodine with starch paper. If resorption is active, a violet color is obtained in from six and one-half to eleven minutes and a blue color in from seven and one-half to fifteen minutes.

Boas states that in dilatation of the stomach the reaction may be delayed two hours, and in cancer as long as eighty-two minutes.

Clinical Value of a Chemical Examination.—The chief points of value relate to the degree of acidity of the stomach contents and may be summed up briefly as follows:

Subacidity, which represents a diminution in the amount of total acidity, and *anacidity*, called *achylia gastrica*, are conditions which consist respectively in diminution and in total lack of hydrochloric acid, and occur in a variety of organic and functional diseases of the stomach and in certain systemic disorders associated with general wasting and with atrophy of the gastric mucous membrane. The secretion of HCl is diminished in chlorosis and pernicious anemia, and the acute infectious diseases, and in chronic wasting diseases, including tuberculosis, diabetes, and Addison's disease; the deficiency being due to functional disturbances of hemic or nervous origin. Subacidity is the rule in the advanced stages of chronic catarrh of the stomach and chronic gastric dyspepsia from irregularities in the diet, and may also be present when the mucous membrane is congested, or affected with acute catarrh with atrophy. Hydrochloric acid is often absent in cancer; but unless the anacidity is found to be constant after repeated examination and two or more additional diagnostic factors of value are present the diagnosis of cancer cannot be made with certainty. In the gastric neuroses the total acidity may be increased or diminished, or may vary at different periods in the same case, as will be more fully explained in the sections devoted to Secretory Neuroses.

Hyperacidity, or *excessive total acidity*, may be due to an excess of hydrochloric acid, when the term *hyperchlorhydria* is used, or to the presence of organic acids which normally exist only in negligible quantities. Hyperchlorhydria, when it is not a secretory neurosis, is characteristic of the early stages of gastric irritation; it is therefore found at times in cases of acute gastritis and in the early stages of gastric dyspepsia, and is practically always present in gastric ulcer. Excess of organic acids—lactic, butyric, and acetic—points to fermentation, as these acids are the result of bacterial activity which is favored by diminution or absence of the normal antiseptic, hydrochloric acid, and by loss of motor power. The organic acids are accordingly found in conditions associated with weakness of the muscular coat, such as dilatation, atony, organic obstruction at the pylorus, and cancer.

Lactic acid is usually present in carcinoma, except, however, when it is derived directly from the food, as after a meal of meat in the form of lactic acid; it is found in appreciable quantities only when there

is loss of motor power with diminution or lack of free hydrochloric acid, and these conditions are found chiefly in cancer. The presence of lactic acid is the most common objective sign in that disease, and is therefore a valuable diagnostic point, the more so as it can be detected before a tumor is palpable; but its absence does not by any means exclude carcinoma. In order to eliminate sarcolactic acid, Boas recommends a test meal consisting of 1 to 2 liters of oatmeal gruel with a little salt to make it palatable, which is to be removed by expression one hour after it has been taken. Since, however, under normal conditions only a minute quantity of lactic acid is found in the stomach contents after an Ewald breakfast, the use of the Boas meal is unnecessary, as considerable quantities only are distinctive.

Diminution of free hydrochloric acid means deficiency of functional activity. As hydrochloric acid directly and indirectly is responsible for the degree of total acidity, the variations in the quantity of free HCl in the stomach contents practically coincide with variations in the total acidity. The clinical value of the remaining chemical tests and investigations need not be explained. They indicate inability of the gastric function to accomplish digestion, but do not point to any special gastric affection. They are of value in distinguishing gastric neuroses from organic disease. In both there are pronounced gastric symptoms; if the examination shows normal digestive powers, a neurosis is indicated.

Gastric Hemorrhage.—Hemorrhage from the stomach, *hematemesis*, or vomiting of blood, is due to an organic lesion or to the effects of acute irritant poisoning. Care must be taken to see that the blood is not from the upper air passages or has been swallowed. If hemorrhage is profuse, the blood may cause irritation of the larynx and provoke paroxysms of coughing. It is often difficult, therefore, to distinguish between hemorrhage from the lungs and hemorrhage from the stomach.

Hematemesis

1. Previous history points to gastric, hepatic, or splenic disease.
2. The blood is brought up by vomiting, prior to which the patient may experience a feeling of giddiness or faintness.
3. The blood is usually clotted, mixed with particles of food, and has an acid reaction. It may be dark, grumous, and fluid.
4. Subsequent to the attack the patient passes tarry stools, and signs of disease of the abdominal viscera may be detected.

Hemoptysis

1. Cough or signs of some pulmonary or cardiac disease precede the hemorrhage in many cases.
2. The blood is coughed up, and is usually preceded by a sensation of tickling in the throat. If vomiting occurs, it follows the coughing.
3. The blood is frothy, bright red in color, alkaline in reaction. If clotted, it is rarely in such large coagula, and mucus may be mixed with it.
4. The cough persists, physical signs of local disease in the chest may usually be detected, and the sputa may be blood-stained for many days. (Osler.)

The hemorrhage may continue within the stomach without exciting vomiting. The general symptoms of hemorrhage may appear first as

pallor, dimness of vision, giddiness, or faintness. The blood is usually acted upon by the gastric juice, and is dark, clotted, and partly digested or mixed with food. Its reaction is acid. In large hemorrhages the blood may be fluid and of a scarlet color; but if retained for any length of time, it is coagulated. When there is only a small amount of blood, the vomited matter has the appearance of coffee-grounds; when the quantity is large and the blood digested the material appears like tar.

Vomiting is usually followed by a characteristic black or tarry stool which may be distinguished from hemorrhage of the intestinal canal below the duodenum by the color of the contained blood. In intestinal hemorrhage the stools are dark red and not necessarily tarry. The dark stools must not be confounded with the same character of stools seen after the administration of iron or bismuth. In some instances a hemorrhage into the stomach may take place from disease of the lower part of the esophagus.

Intermittent or slight continuous hemorrhage as from a gastric or duodenal ulcer may be demonstrated by having a patient swallow a B.B. shot fastened to the end of a braided silk string, the end of which is attached to the teeth and the whole left in place overnight. It is carefully withdrawn and dried the next morning. Reddish-brown discolorations of the string shows the presence of blood. The extreme end of the string will be bile-stained if the shot has passed into the duodenum, as it should.

Causes.—(1) General diseases from changes in the blood, such as scurvy, purpura, hemorrhagic smallpox, yellow fever, acute yellow atrophy of the liver, severe anemia, leukemia, Hodgkin's disease, and pernicious anemia; (2) ulcer of the stomach; (3) cancer of the stomach; (4) ulcer of the duodenum; (5) portal congestion, as in cirrhosis of the liver and other forms of chronic hepatic disease; (6) diseases of the spleen; (7) congestion due to disease of the heart; (8) chronic Bright's disease with atheroma; (9) rupture of an aneurism; (10) vicarious menstruation; (11) Cohen asserts that it occurs in vasomotor ataxia.

CHAPTER XXVII

THE URINE

Inspection of the Urine.—The urine in health is a clear yellow or amber-colored fluid, having a specific gravity of about 1.020, and generally acid in reaction. It contains normally about 45 parts in the 1000 of solid matter, the principal part of which is urea— $21\frac{1}{2}$ parts. The other solids are uric acid and its salts; certain extractives—creatin, creatinin, ammonia, hippuric acid, xanthin, hypoxanthin, sarcin, pigment, etc.; and chlorides, phosphates, sulphates, with their bases, soda, potash, lime, and magnesia.

The volume of urine passed in twenty-four hours is usually about 1200 c.c., but it may fall to 500 c.c. or rise to 2000 without the existence of disease. Women are believed to pass from 150 to 200 c.c. less than men. The volume is diminished when the skin is acting freely, as in warm weather, and when the bowels are loose; and, on the other hand, cold, constipation, and nervous excitement, especially if it induces anxiety and fear, all tend to increase the quantity secreted.

Color.—The color deepens when the urine is concentrated and of a high specific gravity, as after a hearty meal, or exercise, especially in warm weather; and it becomes paler when a large quantity is passed. The color is frequently changed in disease. In fevers the urine soon after being passed is apt to become turbid from the precipitation of urates and the color varies from white, especially in children, to yellow, brown or pink. When the precipitate settles, the supernatant urine may be high colored and clear, or slightly opaque from some suspended matter.

The admixture of pus and chyle gives the urine a milky color. The urine may also be yellowish white and turbid from phosphates, semen, sarcinæ, and bacteria.

The urine is red, reddish brown, or "smoky" in acute nephritis, the color being due to blood. It is bloody in cancer of the kidneys and bladder, and in injuries of the genito-urinary apparatus. When concentrated and containing a large amount of urates the urine is very red and clear. The red color of urine may be due to hemoglobin, constituting hemoglobinuria, or to excess of urobilin, as in scurvy and pernicious anemia. Hemoglobinuria occurs as results of the action of certain poisons, as potassium chlorate; in infectious diseases, as scarlet fever; and in malarial fevers; also in a peculiar disease known as paroxysmal hemoglobinuria. The urine is sometimes chocolate brown when it contains blood. Again, a golden-brown discoloration of the

urine is common in jaundice; frequently the upper layers have a greenish tinge by reflected light. Finally, a red color is produced by the internal administration of logwood and fuchsin.

A yellow color, when opaque, may be due to suspended phosphates and urates. Urine is sometimes of a golden-yellow or saffron color in jaundice, and from the effects of santonin, picric acid, and rhubarb taken internally. A yellow or yellowish-white turbidity may be due also to a mixture of pus and phosphates. The urine usually becomes more or less opaque and yellow if it has undergone alkaline fermentation. Such a change occurs normally within a longer or shorter time after the urine has been passed. It is promoted by heat and exposure to air, and retarded by cold and exclusion from air. Pathologically, in cases of cystitis, the urine when passed is already in a state of alkaline fermentation.

Brown, greenish-brown, or black urine may result from contained bile salts; from indican; from carbolic acid, creosote, and tar used internally or externally; from the internal use of senna, and in cases where there are melanotic tumors. Blue or bluish-green urine may be seen after the ingestion of methylene blue.

Urine is pale usually in proportion as it is copious in quantity. It is paler in those using milk or vegetable diet than in those who eat meats.

Pathologically, pale urine is characteristic of diabetes, chronic Bright's disease, and polyuria of whatever cause. Such urine is also secreted in hysterical attacks, at the crises of febrile diseases, and in anemic conditions.

Quantity.—The volume may be increased, diminished, or unchanged in disease. It is increased principally in three diseases—diabetes mellitus, diabetes insipidus, and in chronic interstitial nephritis. In diabetes mellitus it sometimes exceeds 3200 c.c. The volume may be also increased in cystic degeneration of the kidneys and in double hydronephrosis; in myelomatosis; in certain diseases of the nervous system (tabes, paresis) and as a result of nervous excitement or as a result of increase of arterial tension, copious water drinking, cold weather (inactivity of sweat glands), and after the crises of acute infectious diseases (epicritic polyuria).

The urine is diminished in acute nephritis and in chronic parenchymatous nephritis. All diseases which directly or indirectly impair the force of the circulation lessen the secretion of the urine. Hence, the quantity is diminished in diseases of the heart muscle and in valvular diseases not fully compensated; in emphysema and in chronic bronchitis. It is lessened also in cirrhosis of the liver. In febrile diseases the urine is scanty and high colored and sometimes is almost suppressed (anuria). Copious loss of fluid from diarrhea, hemorrhage, sweating, and vomiting will also decrease the secretion of urine. The urine is sometimes suppressed in acute nephritis, such as follows scarlet fever, and in the final stages of all the organic affections of the kidneys, in shock

and collapse, sometimes after slight operations on the urethra, after the internal administration of drugs the excretion of which occasions violent irritation of the kidney—such as cantharides, turpentine, and even the inhalation of ether, or as a result of an obstruction in the urinary channels.

Specific Gravity.—The average specific gravity of normal urine is about 1.020. It may fall to 1.015 or rise to 1.025, depending upon

the quantity of fluid and food taken, the condition of the atmosphere, especially as regards temperature, and upon mental influences, usually of an emotional character. The specific gravity of the urine is tested by a urinometer graduated for degrees of density between 1.000 and 1.045. As the density of the urine passed at different times during the day varies greatly, the urine for the whole twenty-four hours should be saved and a specimen of this tested.

The method of taking the specific gravity is very simple. A test tube or graduate, having a diameter of about $1\frac{1}{4}$ inches and a length of 6 or 7 inches, is filled with urine to such a point that the lowest part of the urinometer, when slowly inserted, floats clear of the bottom of the tube. The instrument must also float free of the sides of the tube. The specific gravity should then be read off at the level of the eye, the reading being made from the lower portion of the meniscus when the instrument is at rest. Most urinometers are graduated for 60° F., but in ordinary examinations it is not necessary to have the urine exactly at this temperature. In disease the specific gravity varies more widely than in health; it may fall to 1.005 or 1.000 in diabetes insipidus and chronic interstitial nephritis and rise to 1.060 or even higher in diabetes



mellitus. As a rule, to which the urine in diabetes mellitus is the principal exception, the color is an index of the density, pale urine being of low density and high-colored urine of high density.

The specific gravity is increased when the urine is scanty in amount, rarely rising above 1.035, and usually not above 1.028 or 1.030. When the specific gravity rises above 1.035 and the urine is pale in color, the presence of sugar is to be suspected. The specific gravity is lowered in general by the same causes that make the urine copious excepting

saccharine diabetes. Usually, but not always, a urine containing a large amount of albumin is of low density.

Specific Gravity as an Index of the Amount of Solid.—If the last two figures of the specific gravity be doubled the sum will represent the amount of solid matter in 1000 c.c. of urine. The estimate is only approximate, but it is useful.

Reaction.—The reaction of healthy urine is usually acid. The acidity is tested with litmus paper; the blue paper is turned purple or red by an acid, and the red paper is turned blue by an alkali. The acidity of the urine is increased in gout, lithiasis, acute rheumatism, diabetes, chronic Bright's disease, and as the result of the administration of vegetable or mineral acids.

The urine is alkaline because of alkaline fermentation in the bladder in cystitis; from the presence of much blood or pus; from prolonged immersion of the body in a cold bath; in debilitating diseases and anemias and in some cases of gastric neuroses; as the result of the internal administration of alkalis and at times at the height of digestion.

Urinary Sediments.—A white flocculent sediment, composed of epithelium and mucus, occurs normally in most urines after they have stood for some hours.

A dense sediment, varying in color from that of brown sugar to pink or red, consists of amorphous urates. A sediment usually resembling red pepper, but sometimes of a brown color, consists of uric acid. Uric acid is not usually so abundant as is the sediment of amorphous urates; it sinks more rapidly, and is deposited from acid, high-colored urines.

A yellowish or whitish sediment may consist of sodium urate.

A white sediment usually consists of phosphates, associated with which we sometimes find a white sediment consisting of ammonium urate. Such urines are alkaline. A white sediment may be due to uric acid, especially in children.

A yellowish-white sediment may consist of pus, with or without mucus.

A chocolate-brown sediment, occurring in a reddish, smoky urine, consists of blood from the kidneys.

Clots of blood come from the ureters, bladder, and urethra.

Odor.—The odor of normal urine is sometimes spoken of as aromatic, but generally it is sufficiently characteristic to be described best as urinous. When the urine is concentrated, the odor is intensified.

Certain articles of food, such as garlic and asparagus, give the urine characteristic odors. Turpentine, both when taken internally and inhaled, gives to it the odor of violets. The odors of copaiba and cubebs can be detected in the urine of patients who are taking these drugs.

In marked cystitis the natural urinous odor becomes more pungent, and is blended with a strong ammoniacal odor. When much pain is present, and the urine has stood a while, a putrid odor is developed.

In diabetes mellitus the urine has a sweetish, hay-like odor. In

diabetic coma the odor is sometimes like that of chloroform, due to the presence of acetone and diacetic acid in the urine.

Chemical Examination of the Urine.—The chemical examination of the urine, excluding a few simple tests, is for the most part too difficult for ordinary clinical procedure. The exact quantitative estimation of the elimination of the sulphates, phosphates, uric acid, ammonia, nitrogen, etc., without which it is impossible to tell anything about the actual elimination of these urinary constituents, can only be performed by accurate and complicated laboratory methods which are beyond the scope of this work.

Urea.—Urea is the most important final product of nitrogenous disintegration in the body and is approximately 85 per cent. of the entire amount of nitrogen eliminated through the kidneys. Usually the specific gravity of the urine increases in proportion to the amount of urea contained in it. The average daily amount of urea excreted by an adult man between the ages of twenty and forty years is about 30 grams. The urea, like the total volume of the urine, is subject to variations within the limits of health. It is increased after a meal, especially if the latter be rich in nitrogenous food; after copious ingestion of liquids, and by a close atmosphere. On the other hand, fasting, free perspiration, a loose condition of the bowels, and a vegetable or milk diet diminish the amount of urea. Again the absolute quantity increases with the age, the size, and musculature of the person.

The excretion of urea is increased in fever and inflammatory diseases; in diabetes mellitus and insipidus; in malaria, acute leukemia, and in chronic leukemia after x-ray treatment; in purpura hæmorrhagica; in pernicious anemia and after a febrile crisis, especially in pneumonia. It is increased after drinking certain beverages, as coffee, and by many drugs, especially those which act as hepatic stimulants. It is diminished in all forms of nephritis, especially when uremia results; in diseases of the liver, particularly acute yellow atrophy, in acute gout, and chronic rheumatism; in diseases accompanied by emaciation and cachexia; in leprosy, pemphigus, melancholia, imbecility, catalepsy, hysteria, and cholera (Saundby).

There is no satisfactory, rapid method of accurately estimating the elimination of urea. Numerous forms of ureometers have been devised but none are accurate and they are not to be recommended. An exact method has been devised by Folin which may be employed for accurate metabolic studies. (See Simon.)

Chlorides.—Normally 10 to 15 grams of sodium chloride are eliminated in twenty-four hours, the amount depending upon the amount of chlorides in the diet. The absence or presence of chlorides is sometimes of diagnostic value. They are increased in diabetes insipidus, in acute fevers and when absorption of exudates or transudates is going on. They are diminished or absent in pneumonia, in diarrhea, in acute and chronic Bright's disease, in gastric conditions associated with vomiting, and in the febrile stage of the exanthemata. The chlorides can be detected

and roughly estimated with an 8 to 10 per cent. solution of argentic nitrate. A few drops of nitric acid are first added to the urine, to prevent the silver from precipitating phosphoric acid. A single drop of the silver solution will precipitate the chlorine of the silver chloride in a thick white lump, which falls to the bottom of the test-tube, provided the amount present is normal. If, on the other hand, the quantity is diminished, a white cloudiness is produced which renders the whole solution opaque. If no precipitation or cloudiness occurs, the chlorides are absent.

Sulphates.—The daily excretion of sulphuric acid is about $2\frac{1}{2}$ grams, chiefly in the form of sodium and potassium salts—inorganic sulphates. The organic or conjugate sulphates are in combinations with phenol, cresol, indoxyl, skatoxyl, etc. The relation of the first to the second form is as ten is to one. An increase in the total sulphates is seen in the course of acute febrile diseases, in leukemia and in diabetes mellitus. In chronic nephritis and during convalescence from acute fevers the output is decreased. Non-oxidized sulphur bodies (neutral sulphur), representing about 10 to 20 per cent. of the total sulphur eliminated, are of little clinical importance.

Test for Inorganic Sulphates.—To 10 c.c. of urine acidulated with a few drops of hydrochloric acid, 5 c.c. of a 10 per cent. barium chloride solution are added. If the normal amount of sulphates is present, an opaque milkiness develops; if the precipitate is thick and creamy, the sulphates are in excess; if the specimen merely becomes opalescent, they are diminished.

An increase in the percentage of conjugate sulphates indicates retention of the intestinal contents.

Oxalic Acid.—Normally there is eliminated about 10 mgm. of oxalic acid in the twenty-four hours. Although an increase in the amount excreted is present in gastro-intestinal disturbances, in diabetes and in jaundice, the clinical importance of many calcium oxalate crystals lies in the possibility of their forming calculi in some portion of the urinary tract.

Uric Acid.—The normal elimination of uric acid is between 0.2 and 1.8 grams in twenty-four hours, depending (1) upon the diet, as uric acid is an end product of the metabolism of ingested nucleins and purin bases (exogenous uric acid), and (2) upon its formation from the nucleins of the nuclei of the body cells (endogenous uric acid).

The elimination of uric acid is increased in leukemia, in the acute febrile conditions that are associated with a leukocytosis, just before and immediately after the fall of the temperature if it is by crisis or rapid lysis, in gout during and for a short time after an acute exacerbation, in acute articular rheumatism, and in acute yellow atrophy of the liver. Decreased elimination occurs in diabetes, anemia, chlorosis, chronic nephritis, lead poisoning and gout between the exacerbations.

Xanthin Bases.—The purin bases, which are with uric acid the end products of digestion of nucleins (the purin bodies), include xanthin,

hypoxanthin, heteroxanthin, paraxanthin, guanin, and adenin. In general, variations in elimination of these bases correspond to the variations in elimination of uric acid.

Creatin and Creatinin.—An increased elimination of these bodies is observed in acute febrile conditions, a decrease in chronic or wasting diseases, and in the convalescence from acute febrile conditions. They probably represent the end products of katabolism of muscle tissue, and consist of about 4.5 per cent. of the total nitrogen eliminated through the kidneys.

The Phosphates.—The daily elimination of phosphoric acid which is combined with sodium, potassium, ammonium, calcium, and magnesium salts to form phosphates with these bases, varies between 2 and 3 grams, increasing with animal and decreasing with vegetable diet. An increased elimination occurs in diabetes, leukemia, hysteria, and hemorrhagic purpura, while decreased elimination is found in the course of acute infectious diseases and in chronic or wasting diseases. An apparent increase in the phosphates is usually due to increased alkalinity of the urine which causes the precipitation of the phosphates of the alkaline earths and of ammonium-magnesium phosphates. A quantitative estimation of the phosphoric acid of such urines will show no increase in the total amount eliminated.

Ammonia.—About 0.7 gram of ammonium, representing approximately 4.5 per cent. of the total nitrogen, is eliminated in twenty-four hours. An increased elimination occurs in conditions of acidosis (diabetes, starvation, pernicious vomiting of pregnancy), in those conditions that are associated with deficient oxidation (advanced cardiac or pulmonary disease), and in severe disease of the liver parenchyma (acute yellow atrophy, phosphorus-poisoning).

The Albumins.—**QUALITATIVE TESTS FOR SERUM ALBUMIN.**—Serum albumin is of common occurrence but cannot ever be looked upon as a normal constituent of the urine, although its presence by no means indicates disease of the kidney. While it is the ordinary form of albumin present in the urine, other proteins, as serum globulin, nucleo-albumin, histon, albumose, Bence-Jones protein, fibrin, and also hemoglobin, are found at times. The best tests for ordinary albumin (serum albumin) are: boiling with the addition of nitric or acetic acid; overlying cold nitric acid with urine (Heller's test) and the potassium-ferrocyanide tests. Other tests have been devised, *e. g.*, picric acid, trichloroacetic, potassium-mercuric iodide, etc., but for ordinary clinical work the previously mentioned tests are sufficiently delicate and trustworthy. Serum globulin responds to all the following tests for serum albumin. Its differentiation is not difficult, but usually unnecessary.

Boiling and Nitric Acid Test.—A test-tube is filled two-thirds full of filtered urine and the upper third boiled thoroughly, and then a few drops of concentrated nitric acid are added. Any albumin present will be coagulated and appear as a white cloud, contrasting strongly with the clear unboiled urine beneath it. The entire amount of urine in the test-tube

may also be boiled and acidulated and then compared with a control specimen of untreated urine. When there is only a faint trace present it will be overlooked unless the tube be examined against a dark surface in such a way that the daylight falls upon it from above, in front, and preferably a little to one side. Serum globulin is also precipitated by this test. The presence of serum globulin in no way interferes with the test for serum albumin.

If the urine is opaque from amorphous urates, it is unnecessary to filter them out; heat much below boiling will dissolve them, the precipitation of albumin occurring later at a higher temperature.

If the urine is alkaline or faintly acid, phosphates will produce a cloud upon heating the urine; but they are instantly dissolved upon the addition of a few drops of nitric or acetic acid. Concentrated urines should be diluted one-half.

Boiling and Acetic Acid Tests.—This is preferred by many to the preceding test. It is performed in a similar manner, except that a few drops of 25 per cent. acetic acid are added before boiling and again added after the boiling.

The Nitric Acid (Heller's) Test.—This is very simple and accurate in its results. Cold nitric acid is poured into a test-tube to the depth of about an inch. The tube is then inclined to an angle of about 45 degrees and urine allowed to flow gently down upon the acid by trickling along the side of the tube from a pipette or glass tube. At the point of contact of the acid and urine a white, coagulated zone will form in the presence of albumin.

A cloud of urates is sometimes produced and obscures the test. This cloud does not, however, begin at the point of contact and extend upward but at the upper level of the urine and extends downward, and is dissipated by heat.

Patients who are taking copabia or cubebs pass a urine which gives a white zone at the point of contact with cold nitric acid, but heat diminishes the opacity, and the precipitate is soluble in alcohol.

In concentrated urines a secondary ring due to uric acid may form above the junction. It is soluble on gently heating, and does not form when the urine has been diluted. In highly concentrated urine a precipitate of urea nitrate may fall, distinguished by its crystalline nature.

In highly colored urines the urinary pigments form a colored ring at the plane of contact, and in bilious urines the play of colors, as in Gmelin-Heintz's test for bile, is seen.

The urine of patients taking alkaline iodides gives a dense brown ring of iodine, distinguished by adding a few cubic centimeters of chloroform and mixing. A violet tinge is imparted to the liquid.

Albumoses are precipitated (as well as all forms of albumin), distinguished by partial or complete disappearance upon heating, to reappear upon cooling.

This estimation, as already stated, is not absolutely accurate. Nevertheless, if used systematically, and always in the same way, relative values may be obtained, and these are the most important in watching the progress of a case, as they give positive information regarding an increase or diminution of the amount of albumin in the urine. It scarcely need be said that the urine tested must be a portion of the whole twenty-four hours' urine.

A solution of phosphotungstic acid, 1.5 grams; hydrochloric acid, 5 c.c.; and ethyl alcohol, 95 c.c., is also employed in a similar manner, and it has the added advantages that the urine requires no dilution and the readings are uninfluenced by the temperature.

ALBUMINURIA.—Albuminuria is not indicative of disease of any one organ nor does it point to any general pathological condition. It occurs:

1. In diseases of the kidney: as acute and chronic Bright's disease, amyloid disease, tuberculosis, cancer, abscess, and obstruction of the renal arteries or veins.

2. In disturbances of the circulation: diseases of the heart and chronic pulmonary diseases, as emphysema; in local circulatory obstruction as pregnancy and abdominal tumors; in passive congestion due to great weakness and in Graves' disease.

3. In febrile and inflammatory diseases: as in the eruptive and infectious fevers, in rheumatism, diphtheria, pneumonia, and gout.

4. In blood diseases: purpura, leukemia, scurvy, and anemia.

5. From the poisonous action of drugs: lead, turpentine, and after inhalations of ether and chloroform.

6. In nervous disorders: concussion of the brain and cerebral hemorrhage, epilepsy, tetanus, and delirium tremens.

7. In any condition obstructing the outflow of urine: calculus, tumor.

8. In local extrarenal affections: pyelitis, cystitis, urethritis, and vaginitis, depending upon the presence of pus or blood in the urine (spurious or accidental albumin).

9. As a functional disturbance: in young persons, particularly of the male sex, after exercise, a special diet, or a cold bath. Albumin may be found after rising in the morning, or early after dinner, or toward evening. On account of its occurring only at certain times it has been called "cyclical" or "intermittent;" and because there is no evident disease present it is occasionally spoken of as "physiological" albuminuria; and because of its appearing after the patient has been on his feet and disappearing when in bed, it is called "postural" or "orthostatic" albuminuria. Other self-explanatory synonyms are "dietetic" or "digestive" and "adolescent albuminuria."

It is conceded that there may be albuminuria of renal origin without renal disease, but the diagnosis must be by exclusion, and can be reached safely only after extended observation. The most important elements in the diagnosis are: the age of the patient, unimpaired general health, a specific gravity of the urine normal or above normal, the fact that the albuminuria is influenced by diet and exercise, and that it tends to disappear under suitable regimen. The prognosis is favorable.

of red cells in the urine in small quantities is usually noted upon microscopic examination of the centrifugated sediment. Failure to find them in a microscopic examination of a fresh urine discolored by blood-coloring matter or showing the presence of hemoglobin by chemical tests, makes the diagnosis of hemoglobinuria.

Blood occurs in the urine from (1) diseases of the kidney and urinary passages, among which are Bright's disease, acute congestion of the kidney, renal calculus, cancer, tuberculosis; from ureteritis, cystitis, urethritis, and injuries; (2) from general diseases, such as the eruptive and intermittent fevers, scurvy, purpura, hemophilia, leukocythemia, and cholera; (3) from adjacent organs, as in menstruation and hemorrhage from the uterus; (4) from the toxic action of drugs—cantharides, turpentine, and other violent irritants of the kidney; (5) vicariously—occasionally menstruation fails to occur and hematuria replaces it.

Hemoglobin.—Hemoglobin, while present whenever there is hematuria, may appear independently in the urine in any condition causing extensive hemolysis (toxic hemoglobinuria). It may appear without cause in distinct paroxysms, or it may be the manifestation of an epidemic infectious disease of the newborn. *Toxic hemoglobinuria* is found in grave infectious diseases, such as yellow fever, scarlet fever, typhoid fever, syphilis, etc.; as the result of toxic action of drugs, such as carbolic acid; after extensive burns; and as a result of injections of the blood serum of one species of animal into a different species. Craig believes the hemoglobinuria that has been variously attributed to a malarial toxemia or to the action of quinine is a disease *sui generis*.

Paroxysmal Hemoglobinuria.—This is most frequent in adults; it may be excited by a cold bath or exposure to cold, or by exertion. It is sometimes associated with Raynaud's disease. The attacks come on suddenly, often preceded by chills. Sometimes fever accompanies the disease. Pain in the loins is sometimes severe. The paroxysm may last a day or two, or two or three paroxysms may occur in the course of twenty-four hours.

Epidemic Hemoglobinuria.—This disease develops most frequently in the third or fourth day of life, and may attack many children in a maternity hospital. It is characterized by hemoglobinuria, vomiting, purging, fever, and jaundice, going on to rapid collapse and death in a short time.

Test for Hemoglobin.—Add excess of sodium hydrate to a small portion of urine and sediment. Boil. On cooling a red precipitate appears, consisting of phosphates and adhering blood pigment. Its presence can be definitely determined with the spectroscope.

Pus.—Pus is found in the urine whenever there is suppuration or a catarrhal condition of the genito-urinary tract. Hence it occurs in abscess of the kidney, pyonephrosis, pyelitis, tuberculosis, cystitis, gonorrhea, leucorrhea, etc. It is relatively common in women, from a catarrhal condition of the vulva and vaginal mucous membrane, and

is therefore of less significance than in men. Urine containing much pus is slightly albuminous; but frequently pus cells are found in urine which gives no reaction for albumin.

Sugar (Glucose).—Sugar, though present in normal urines in minute amounts, cannot be detected by the ordinary chemical tests. A persistent glycosuria as determined by the ordinary tests is found in diabetes mellitus and in cerebral lesions involving the floor of the fourth ventricle.

A transitory glycosuria may occur in the course of cerebral affections, acute febrile diseases, Graves' disease, general paresis, acute pancreatitis and certain disorders of the pituitary body, and after ingestion of phloridzin, or certain poisons, as chloral hydrate, alcohol, and morphine.

A transitory "digestive glycosuria" occurring after the ingestion of large amounts of sugar (100 grams of glucose) has been observed in a certain percentage of patients suffering from diseases of the liver, Graves' disease, general paresis, acute febrile affections, hyperpituitarism, alcoholism, psoriasis, and in pregnancy.

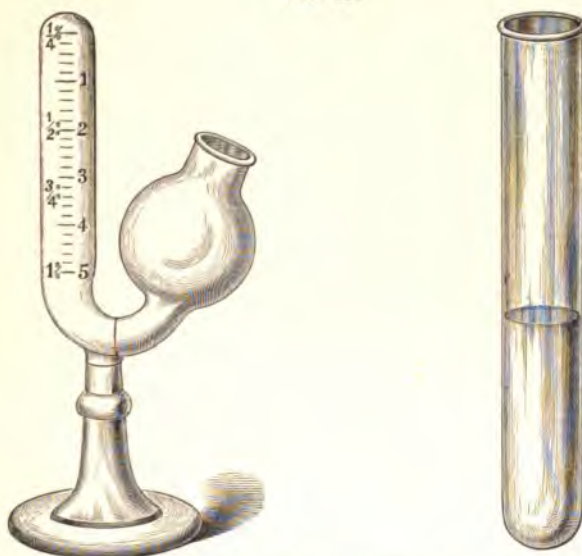
Test for Sugar.—Fehling's Test.—Two solutions are made up: (A) 34.64 grams of crystallized cupric sulphate, dissolved in 500 c.c. of distilled water; (B) 173 grams of sodium and potassium tartrate (Rochelle salt) and 50 grams of sodium hydrate, dissolved in 500 c.c. of distilled water. 1 c.c. of equal parts of these two solutions is reduced by 0.005 grams of sugar. When ready to test the urine, equal parts of the two solutions are placed in a test-tube and diluted with four times the volume of water. The solution is boiled and a small quantity of urine added. The solution is again heated—not boiled, however—and if sugar is present a yellowish-orange precipitate of copper hydroxide or of red cupric oxide forms. If negative, repeated small portions of urine may be added to the solution until an amount of urine equivalent to the volume of copper solution has been added. Absence of precipitate when this much urine has been tested shows absence of sugar.

Certain precautions are necessary in the application of this test: (1) any albumin present must be removed by boiling and filtration; (2) boiling the reagent first in a test-tube is a test of its stability; if a precipitate occurs, the solution is unfit for use; (3) when the earthy phosphates are abundant, it is well to get rid of them by adding a small quantity of sodium hydroxide and filtering before applying the sugar test; (4) reduction of copper may occur from the presence of uric acid, creatinin, nucleo-albumin, bile pigment, alkapton bodies, lactose, or after the ingestion of sulphuric acid, chloral, or sulphonal; this reduction can usually be obviated by not boiling the solution, merely heating it almost to the boiling point; (5) a greenish discoloration and turbidity of the solution after standing for a time is not due to sugar but is probably the result of one of the above reducing substances.

The Fermentation Test.—This is based upon the fact that sugar upon fermentation with yeast breaks up into alcohol and carbon dioxide.

It is both a reliable and a very delicate test for sugar. A piece of yeast cake the size of a pea is added to a fermentation-tube full of urine without air bubbles at the top. If sugar is present bubbles of carbon dioxide collect at the upper part of the closed tube after standing twenty-four hours in a temperature of about 37°C . At the same time it is best to make two control tubes, one containing the urine without yeast, and the other normal urine with yeast.

FIG. 129



Einhorn's saccharimeter.

The Phenylhydrazin Test (V. Jaksch's Method).—About 0.5 gram of phenylhydrazin hydrochloride and about 1 gram of sodium acetate are added to 10 c.c. of urine in a test-tube. The tube is kept for fifteen to twenty minutes in boiling water and then put in a vessel of cold water. When a large amount of sugar is present, a deposit of yellow needle-like crystals is visible to the naked eye; but when only a small amount is present, the sediment must be examined under the microscope. The crystals appear singly, or in sheaves and fine radii. Yellow plates and brown balls do not indicate sugar.

Nylander's Test.—2 grams of bismuth subnitrate, 4 grams of Rochelle salt, and 10 grams of sodium hydrate are added to 90 c.c. of water, the whole is then boiled, filtered and kept for future use in a dark bottle. To test for sugar, remove any albumin present in the urine by boiling and filtering. Add to 10 c.c. of the filtered urine 1 c.c. of the reagent and slowly boil. In the presence of sugar the fluid becomes dark brown and finally black in color. A brown coloration

Glycuronic acid, derived from glucose, occurs in urine after the administration of chloral, butyl-choral, nitrobenzol, camphor, curare, morphine, chloroform, fusel oil; it reduces Fehling's solution, is dextro-rotatory, and does not ferment.

β -oxybutyric acid occurs in severe diabetic cases, usually in association with diacetic acid and acetone; it has strong levorotatory action, and may interfere with the polariscope test. If a urine is strongly levorotatory after fermentation, this substance is probably present.

Acetone.—An excess of acetone may be eliminated in the following disorders: (1) diabetes; (2) in cancer of the stomach, independent of starvation; (3) in starvation; (4) in certain psychoses; (5) in pernicious vomiting of pregnancy; (6) in derangements of digestion; (7) in continued fevers; (8) after chloroform narcosis; (9) acute phosphorus poisoning. In diabetes, acetone indicates a severe acidosis. *Lieber's test* for acetone is as follows: to several cubic centimeters of distilled urine a few drops of iodo-potassic iodide (Lugol's solution) and sodium hydroxide are added; if acetone is in excess, precipitation of iodoform takes place, which may be recognized by its odor upon warming the solution or by microscopic examination when the iodoform crystals will be seen.

Legal's Test.—A few drops of concentrated watery sodium nitroprusside is added to the urine, which is rendered alkaline with sodium hydrate. In the presence of acetone the red color changes to violet and finally to deep purple.

Diacetic Acid.—This is found in the urine in diabetes, in fevers, and in the other conditions in which acetone is found. Its significance is practically the same, but to a greater degree. It is common with children in fever. It is of grave significance in the urine of adults. Coma usually follows its occurrence in the urine during fevers, and diabetes. Test: a concentrated solution of ferric chloride is cautiously added to the urine; if a precipitate is formed it should be removed by filtration and more chloride added to the filtrate. If diacetic acid be present, the liquid will become claret red in color.

Other acids occur pathologically in the urine, such as *lactic acid*, which is present in phosphorus poisoning, and acute yellow atrophy, as are *tyrosin*, *leucin*, and *glycocol* (amino acids). *Volatile fatty acids* are found to be increased in suppurative and hemorrhagic conditions within the body.

Urinary Pigments.—The normal color of the urine is due to the presence of urochrome. Very slight variations in the urine may cause the appearance of uroerythrin, the pigment which gives the reddish pink color to the urate sediments. The chromogen indican is normally present in small amounts and is of clinical interest because of the marked increase in its formation in certain pathological conditions. Abnormal pigments of clinical importance include the bile pigments, blood pigments, melanin, and alkapton bodies. The urine may also be discolored by the action of certain drugs. Thus after the ingestion of indigo or methylene blue the urine becomes blue in color. Santonin,

rhubarb, and senna give a bright yellow color to the urine, which in the presence of an alkaline becomes red. Carbolic acid and salicylic acid may impart a brownish tint to the urine.

INDICAN.—This substance is detected by several methods. *Jaffe's test*: equal volumes of concentrated hydrochloric acid and urine are mixed. By means of a glass pipette a strong solution of sodium hydrochlorite is dropped into the fluid. The mixture is well shaken with several cubic centimeters of chloroform. An indigo-blue color is produced in the chloroform if indican be present. The hypochlorite must not be added in excess.

Obermeyer's Test.—2 gms. of pure ferric chloride are dissolved in 1000 c.c. of concentrated hydrochloric acid; 5 c.c. of this reagent are added to an equal amount of urine and a small amount of chloroform added. The whole is then well shaken. Normally the chloroform is faintly blue or colorless. An increased quantity of indican is shown by a dark blue color. Indican is increased by meat diet; an increase which under other circumstances is pathological. Its presence in health is a sign of intestinal putrefaction. It may accompany decomposition of albumin in cavities. It is present in empyema and in puerperal peritonitis. Indican is increased in acute diarrhea and in intestinal tuberculosis. V. Jaksch states that large quantities of indican in the urine imply that abundant albuminous putrefaction or putrid suppuration is in progress in the body. It must not be forgotten that indicanuria will often arise in constipation in association with disease elsewhere, as in hyperchlorhydria.

BILE PIGMENTS.—Bilirubin occurs in freshly passed urine in cases of jaundice (*q. v.*). Biliviridin, biliprasin and bilifuscin, oxidation products of bilirubin, may be found in icteric urines that have stood for some time. The following tests for bile pigments may be employed:

Gmelin's Test.—A small quantity of nitric acid which has been exposed to the air, is put in a test-tube and then gently overlaid with urine. If bile pigment is present, a series of colors appear at the junction of the two fluids—green, blue, violet, and yellow. The green color is most typical.

Rosenbach's Modification.—About 200 c.c. of urine are allowed to flow through pure white filter paper, and then a drop of nitric acid is placed upon the paper saturated with the urine. The colors appear as before described.

Smith's Test.—5 c.c. of urine in a test-tube is overlaid with 2 c.c. of a solution of tincture of iodine, one part; alcohol, nine parts. At the point of contact a brilliant green ring is seen if bilirubin is present.

The tests for bile acids, usually found in the urine whenever bile pigments are present are either too elaborate or too unsatisfactory for clinical use.

PATHOLOGICAL UROBILIN—Differentiated from urochrome (normal urobilin) by the spectroscope, this pigment is usually found in febrile urines and in the urine of hepatic cirrhosis. Less frequent is its occur-

rence in the urine of pernicious anemia, carcinoma, scurvy, hemophilia, cerebral hemorrhage, chronic malaria, Addison's disease, and secondary syphilis.

Schlesinger's Test.—10 c.c. of 1 per cent. solution of zinc acetate in absolute alcohol are added to an equal amount of urine, well shaken and filtered. A distinct fluorescence of the filtrate shows the presence of urobilin.

Gerhardt's Test.—Extract 10 c.c. of urine with chloroform by shaking. Add a few drops of KI + I solution (Lugol's solution) to the extract. Upon the addition of dilute sodium hydroxide, the chloroform extract turns a yellowish brown and shows a beautiful greenish fluorescence, more intense than with normal urobilin.

PHENOL.—Phenol, normally present in minute amounts in the urine, is increased whenever putrefactive processes occur in the body. An excess can be demonstrated by the presence of turbidity, or precipitation upon boiling 10 c.c. of urine with 3 c.c. of nitric acid and then adding bromine water when cool.

HEMATOPORPHYRIN.—This is a constituent of the urine derived from the blood, occurring in traces in every urine. It is a form of hematin freed from iron. Nakarai thinks that the occurrence of marked hematoporphyrinuria is constant in lead and trional and sulphonal poisoning and occurs with some degree of frequency in intestinal hemorrhage. Increased hematoporphyrin has also been demonstrated occasionally in the urine in other diseases, notably hepatic disease. The other blood pigments have already been discussed.

FIG. 130



Crystals of cholesterol. (Original.)

CHOLESTERIN.—This occurs at times in fatty degeneration of the kidneys, jaundice, chyluria, diabetes.

MELANIN.—Melanin is held in solution or suspended in small granules. The urine is dark in color, and blackens intensely when an excess of a concentrated solution of ferric chloride is added to it. Melanin is chiefly found in cases of melanotic tumors.

ALKAPTON.—The substance in the urine which has been identified as alkapton is known as homogentismic acid. It reduces copper, and its occurrence is of interest because the presence of the substance has led to the diagnosis of glycosuria in many instances, in consequence of which persons have been refused life insurance. The urine containing this substance deepens in color on exposure to the air. It is of a peculiar aromatic odor, and reduces cupric salts rapidly. There is, however, no reaction to the fermentation test, to the bismuth test, or to phenylhydrazin, and no deviation of polarized light. The urine does not contain bile pigments. It is of a normal specific gravity, and becomes very dark on the addition of an alkali, or of a temporary bluish-green color with ferric chloride. Ammoniacal nitrate of silver when added to the urine is instantaneously reduced with a deposit of metallic silver.

Alkaptonuria.—This is usually a congenital anomaly of metabolism. Several members of the same family may have it. The only characteristic symptom of the condition is irregular blackening of the cartilaginous and fibrous tissues and pigmentation of the skin (ochronosis).

Diazo Reaction.—Two solutions are necessary for this test: (1) sulphanilic acid, 1 gram; concentrated hydrochloric acid, 50 c.c.; distilled water, 1000 c.c.; (2) sodium nitrite, 1 gram; distilled water, 200 c.c. Fifty parts of No. 1 solution are mixed with one part of No. 2. Equal parts of urine and the mixed reagent are put in a test-tube and then rendered alkaline with ammonia water. A positive reaction is shown by a red coloration of the fluid and of the foam when well shaken. The reaction is practically always positive in typhoid fever, measles, and tuberculosis. In the latter disease it is less likely to be present in the chronic than in the acute forms, and if present usually indicates an extensive or rapidly progressing lesion. The reaction is often present in scarlet fever, pneumonia, erysipelas, diphtheria, septicemia, and pyemia.

Microscopic Examination of the Urine.—Microscopic examination of the urine is concerned with the sediments, and these are conveniently divided into the organized and the unorganized.

The organized deposits in the urine are blood, pus, mucus, epithelium, casts, spermatozoa, tumor fragments, and animal and vegetable parasites.

The unorganized deposits are uric acid and its compounds, oxalate and carbonate of lime, phosphates, sulphates, leucin and tyrosin, cystin, cholesterin, hippuric acid, bilirubin (hematoidin) crystals, etc. Normal urine forms a slight sediment, consisting of epithelium from different parts of the genito-urinary tract, principally from the bladder in males and from the vagina and bladder in females. There are also crystals of the different urinary salts, sometimes mucus, and a few white blood

cells, and if the urine has stood a while, especially if it is alkaline, more or less bacteria. The sediment may be collected by allowing it to settle for several hours in the bottom of a bottle or conical glass, or better still, by centrifuging a portion of the specimen for a few minutes. A very small whitish sediment or cloud only may be found at the bottom of the vessel. If calcium oxalate is present, a small, filmy, whitish

FIG. 131



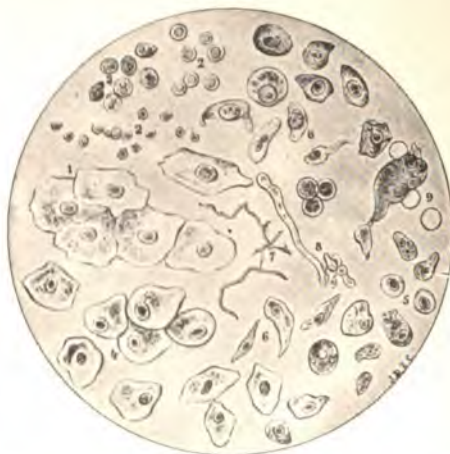
Extraneous matters found in urine. *a*, cotton fibers; *b*, flax fibers; *c*, hairs; *d*, air bubbles; *e*, oil globules; *f*, wheat starch; *g*, potato starch; *h*, rice starch granules; *i*, *i*, *i* vegetable tissue; *k*, muscular tissue; *l*, feathers.

sediment is seen. The sediment of amorphous urates is pinkish, fawn, or salmon color. Uric acid appears as a "brick-dust" sediment. Pus produces a heavy yellowish sediment; phosphates a heavy white sediment, which is sometimes yellowish white from admixture with leukocytes. Blood in small quantities produces a rather characteristic, brownish deposit. Large amounts of blood appear as reddish coagulate

at the bottom of the tube. The sediment is withdrawn from the very bottom of the vessel by capillarity through a straight glass pipette, the end of which is blunted. A few drops of the sediment are drawn into the tube and then spread in a thin layer over the surface of a clean slide, which is examined, without applying a cover-slip, with the low power, the greater part of the light being cut off, and then examined more carefully, in the case of any doubt, with the high power.

Organized Sediments.—**BLOOD.**—If the blood comes from the kidney it is usually intimately mixed with the urine, which remains of a red or reddish-brown color, and contains possibly tube casts and renal epithelium (Fig. 132).

FIG. 132



Cellular elements from the urine. 1, squamous epithelium; 2, red-blood corpuscles; 3, polynuclear leukocytes; 4, transitional cells; 5, epithelium from the kidneys; 6, epithelium from the bladder; 7, *Micrococcus ureæ*; 8, yeast fungi.

Sometimes blood coagulates in the ureters and long cylindrical plugs are passed, causing symptoms resembling those of renal colic. When blood comes from the bladder or neck of the bladder (fissure) frequent micturition, acute pain, and tenesmus are observed, and the blood is not intimately mixed with the urine. When the hemorrhage is from the neck of the bladder it often occurs as a few drops at the end of micturition, accompanied with great pain and a sense of faintness. Blood in the first part of the urine, the latter part of which is clear, denotes some lesion in the urethra. Idiopathic hematuria is said to occur at times. In any case of hematuria without clinical symptoms of disease in the genito-urinary tract recourse should be had to the cystoscope, to ureteral catheterization and to the x-ray, before making the diagnosis of such a disorder, as the hemorrhage may come from a new growth of the bladder, from renal tuberculosis, or some other condition without other clinical manifestations referable to the tract.

Blood cells when unaltered are unmistakable on account of their well-known biconcave appearance. When they have lost their coloring matter they appear as circular, very pale disks, with extremely faint outline and feeble refractive power. Absence of a nucleus serves to distinguish them from leukocytes and from yeast spores; the latter, moreover, are often oval in shape. They are less likely to be confounded with the ovoid and circular shapes of calcium oxalate crystals because the latter are not common, and are seen usually in their more common forms as octahedral and dumb-bell in the same urine.

Pus.—Leukocytes appear normally in the urine in small numbers. A marked increase, however, is pathological and is indicative of disease somewhere in the urinary tract. The pus corpuscle is a refractive, spherical, granular cell, usually somewhat larger than red-blood cells. In concentrated urines the pus cell is small; in diluted urine, or urine to which water has been added, it swells sometimes to twice its original size. At the same time it becomes less granular, and two, three, or four nuclei may appear. The addition of acetic acid also causes it to swell, and brings out the nuclei more distinctly and rapidly. Sometimes the pus cells are discrete, sometimes in small clumps, and sometimes nothing but a dense mass of pus cells appears in the field of the microscope.

The source of the increased number of leukocytes must be a matter of inference from the general character of the urine. If red-blood cells are also present, the probability of finding many white-blood cells is increased. Urine containing pus from the kidney is usually acid, whereas in cystitis it almost always contains phosphates, mucus, abundant bacteria, and is alkaline in reaction, although cystitis due to *B. coli communis* or *B. typhosus* is regularly associated with acid urine. Again, pus from the kidney or the pelvis of the kidney is apt to vary greatly in amounts or be discharged intermittently; and the urine when filtered free from pus cells is usually still albuminous. Renal epithelium and casts may also be found.

CASTS.—Tube casts or urinary cylindroids are terms applied to moulds of a coagulated serum-like substance formed in the uriniferous tubules, the result of alteration of the kidney substance. They are the most important of the urinary deposits, and they may be so numerous that nearly every field contains a dozen or more; or they may be very few, not more than one or two being found on a slide. When but few casts are present, several slides should be examined with the low power. While tube casts always indicate some irritation of the renal epithelium and hence are always found in nephritis, nevertheless the occasional presence of casts in the urine does not necessarily signify nephritis, as the irritation of the renal epithelium may be due to transitory conditions such as vigorous exercise, cold baths, anemia, infections, intoxications or passive congestion, which may subside without any injury to the renal parenchyma. Several varieties of casts are found.

1. *Hyaline casts* are clear, translucent bodies, which reflect light so slightly that they are easily overlooked. They have well-defined margins, the ends being frequently rounded; they are rarely very long,

FIG. 133



Hyaline casts from a case of acute nephritis: 1, plain hyaline cast; 2, granular deposit on hyaline cast; 3, cellular deposit (blood and epithelium).

FIG. 134



Granular casts.

and are straight or but slightly bent. They are rarely equally translucent throughout; at some parts more or less granulation will generally be found. They vary in diameter from that of a white blood cell to those six or eight times as wide.

2. *Granular casts* are hyaline casts which appear granular either from some deposit on their surface or from a granular change of the

cast itself. When the granulation does not interfere with the translucency the casts are described as "pale" or "slightly" granular; and when they become very dark, so as to resemble closely a blood-cast, they are called "dark" or "opaque" granular casts. At times brown or dark granular casts are seen which show no trace of a hyaline matrix and which are probably composed of closely packed degenerated epithelium cells in cast form.

3. *Waxy casts* appear to the eye to be more solid in structure than the hyaline casts; they also appear more cylindrical in form, are more or less yellow in color, and are apt to be larger than hyaline casts.

4. *Fatty casts* are hyaline or faintly granular casts on which are deposited in spots, minute oil drops. If the oil drops are very abundant, they are sometimes called "oil casts."

FIG. 135



Fatty casts from a case of chronic parenchymatous nephritis.

5. *Blood casts* are either made up of a mass of blood cells pressed into a cylindrical shape, or more frequently they are hyaline casts studded with blood cells.

6. *Epithelial casts* sometimes seem to be composed entirely of epithelial cells closely packed. Such casts are relatively rare, but very striking. Ordinarily, just as in the case of blood casts, an epithelial cast consists of a hyaline cast more or less covered with renal epithelium.

7. *Pus casts* are usually described as more or less regularly occurring in purulent kidney affections.

8. *Cylindroids* are very common. In general appearance they resemble hyaline casts; but they are apt to be much longer, bent, twisted, or split, and to have a striated or finely ribbed appearance on close examination. Moreover, the diameter of the cast frequently varies greatly at different points; sometimes it appears constricted in

several places, and in most cases one end tapers into a thread. Often cylindroids consist of fine, narrow, ribbon-like threads.

Significance of Casts.—It is impossible to diagnosticate the type of nephritis from the character and number of casts. In general it may be said that hyaline casts when alone, indicate temporary disturbance of the renal epithelium; in association with granular and fatty casts, destruction of the renal cells; epithelial and blood casts point to an acute process or exacerbation; pus casts to a suppurative process; waxy casts or dark granular casts usually indicate a chronic process. In regard to the number of casts, a few transitory casts usually mean a transitory irritation of the parenchyma; a few casts continually excreted, usually a chronic interstitial nephritis; many casts, an acute or subacute process; and finally numerous casts may be discharged at irregular intervals,

FIG. 136



Cylindroids.

the so-called "cast showers," without indicating any particular change in the lesion already present. Cylindruria is usually associated with albuminuria, although one may occur without the presence of the other. Cylindroids are usually found in connection with hyaline casts and possibly have the same significance.

SPERMATOCYTES.—Spermatozoa are usually recognized by their tadpole shape and by the vibratile motion of their long, delicate tails. They are found in the urine after sexual intercourse or nocturnal emissions.

EPITHELIUM.—Epithelium from the kidney, bladder, and genito-urinary passages occurs in the urine. Epithelial deposits in male urine are very scanty unless there is some disease of the kidney or bladder, or a catarrhal condition of the prostatic urethra, such as is left after an old gonorrhea. On the other, a considerable number of large, flat,

epithelial cells may be normally present in the urine of women, being derived principally from the vagina.

In general, three types of epithelial cells may occur in the urine: (1) round cells, somewhat larger than a leukocyte, with relatively large nucleus, derived from the uriniferous tubules or deep layers of the mucous membrane of the renal pelvis and from the male urethra; (2) flat cells, large polygonal uninuclear cells, derived from the ureter, bladder, or genitalia; (3) conical and caudate cells, derived from the renal pelvis.

The study of the morphological characteristics of the urinary epithelium yields knowledge that at the best merely suggests the location of a lesion in the urinary tract. Thus round cells attached to casts suggest an inflammatory kidney lesion, while round cells associated with conical cells and pus cells, with only a trace of albumin and an absence of casts, probably denotes a pyelitis.

FAT.—Oil is found in the urine in fatty degeneration of the kidney and its epithelium, and occasionally in the urine of those who are taking cod-liver oil, and in diseases of the pancreas and liver. It is also found at times in cachectic conditions, in phosphorus-poisoning, and in diabetes mellitus, as well as in chyluria and after fractures of bones. The urine is turbid, but clears when agitated with ether. The fat can be recognized by its strongly refractive properties.

Staining for Fat.—A saturated solution of Sudan III in 96 per cent. alcohol is employed. Equal parts of this solution and 90 per cent. alcohol are added to the urine. In ordinary sediments the fat droplets in casts stain a scarlet red. Large fat droplets take a bright red, and small droplets a yellow or orange color.

CHYLE.—Chyluria is a more or less milky condition of the urine, due to the presence of fat, which probably gains entrance to some part of the urinary tract by rupture of the lymphatic vessels. Fat and albumin appear at the same time in parasitic chyluria, which is due to *Filaria*, whose embryos obstruct the lymphatics, causing lymph varices, which may rupture into the bladder. They recur at long intervals. Red and white blood corpuscles are also found in small amounts. The urine coagulates on standing or gelatinizes.

PARASITES.—*Animal Parasites.*—The occurrence of animal parasites in the urine is usually rare. *Filaria* embryos and the ova of *Bilharzia* may occur in the urine of patients affected by these parasites. *Echinococcus* hooklets and particles of the cyst walls as well as *Trichomona vaginalis* are found in the urine when these parasites infest the genito-urinary tract. The occurrence of other parasites in the urine is so rare as to be of little clinical importance.

Vegetable Parasites.—Normal urine contains no microorganisms, but as the urethra is not sterile bacteria are always present in the urine as passed. Urine exposed to the air becomes contaminated in a short time not only by bacteria, but by yeasts and moulds also, and may undergo more or less rapid ammoniacal fermentation, largely the result

soluble in hydrochloric acid. They are found normally after the administration of large amounts of sodium bicarbonate or vegetable acid salts and after eating rhubarb, asparagus, tomatoes, and oranges. In gastro-intestinal neuroses, diabetes, psoriasis, psoriasis, and in cases of so-called neurasthenia, they frequently appear in large numbers in the urine.

Fig. 126



Uric acid.

Fig. 127



Uric acid crystals are found in the urine of patients with gout, rheumatism, and other uric acid disorders. They are usually found in the sediment of the urine, and are often associated with other urinary solids.

Uric acid.—Uric acid crystals appear as large, refractive, reddish-brown, six-sided plates which are soluble in ammonia and insoluble in water or acetic acid. They are rarely found, though, in the urine of a normal person.

Uric acid needles.—Long, narrow, needle-shaped crystals, insoluble in ammonia and acetic acid. They are without clinical significance.

Uric acid is usually described as being found in the urine of patients with gout, rheumatism, and other uric acid disorders.

form of spheres, which refract light strongly and have a radiating arrangement (Fig. 139). Tyrosin appears in the form of long needle-like crystals arranged in bundles or sheaves. Leucin and tyrosin are found consistently in acute yellow atrophy of the liver and in many cases of acute phosphorus poisoning. Minute quantities are present occasionally in catarrhal jaundice, Weil's disease, cholelithiasis, cancer and cirrhosis of the liver, nephritis, gout, diabetes, typhoid fever. To demonstrate these crystals either evaporate the urine to a small quantity and examine the sediment, or perform the following precipitation procedure. Add an excess of basic plumbic acetate; filter; pass hydrogen sulphide through the filtrate; again filter and then evaporate the filtrate almost to dryness; shake with absolute alcohol; extract the insoluble residue with alcohol containing a little ammonium. Leucin and tyrosin are precipitated out when this extract is concentrated to a small amount.

FIG. 140



Tyrosin crystals. (Original.)

Soaps of lime and magnesium are said to resemble tyrosin crystals, but may be differentiated by the fact that a hot solution of tyrosin, acidified with 1 per cent. solution of acetic acid and sodium nitrite added, turns a brilliant red.

Hippuric Acid.—Fine needles or transparent colorless rhombic prisms, frequently arranged in clusters, soluble in alcohol and insoluble in hydrochloric acid, have been seen in febrile and diabetic urines. They may occur after administration of benzoic or salicylic acid, or the ingestion of blueberries or cranberries.

Bilirubin (Hematoidin) Crystals.—These crystals appear in the form of brilliant yellow rhomboids or needles. A drop of nitric acid added to the slide will give a greenish play of colors to the sediment. The crystals are found at times in nephritis, acute diseases of the liver, or
* the bladder.

SEDIMENTS IN ALKALINE URINES.—*Ammoniomagnesium Phosphates*, or triple phosphates, are colorless, various sized, prismatic crystals which resemble coffin-lids. Long, feathery crystals in an amorphous form are also occasionally seen. These crystals are soluble in acetic acid.

FIG. 141



Triple phosphates. (Original.)

FIG. 142



Ammonium urate. (Original.)

Amorphous Calcium and Magnesium Phosphates.—These are colorless, transparent, granular bodies, resembling amorphous calcium phosphate. They are soluble in acetic acid, and are unaffected by heat.

Ammonium Urate.—These crystals have a characteristic thorn-apple form, appearing as brownish or black spherical bodies with sharp projections (thorn-apple crystals).

Neutral Calcium Phosphates.—Colorless, slender, pyramidal crystals united to form rosettes or star-like figures, appearing in neutral as well as alkaline urines, and are soluble in acetic acid.

FIG. 143



Calcium phosphate crystals. (Original.)

Neutral Magnesium Phosphate.—Long, colorless, highly refractive plates with irregular edges, soluble in acetic acid, may very rarely be present in neutral or alkaline urines.

Calcium Carbonate.—Amorphous granules, often arranged in spheroidal masses, are recognized as crystals of calcium carbonate by the fact that they give off CO_2 when treated with acetic acid.

CHAPTER XXVIII

THE FECES

General Considerations and Macroscopic Appearance.—Quantity

The quantity of feces varies with the quantity and nature of the food, a diet rich in protein leaves but little residue, while one containing much starch and vegetable food leaves considerable residue. In any disease that prevents the absorption of digested food or causes an increase in the fluid contents of the intestine, the amount of feces will be increased. In health about 140 to 200 grams of fecal material are voided in twenty-four hours.

Form. The form and consistence of healthy stools vary somewhat, depending upon the amount of liquid they contain. They are commonly cylindrical and firm or mushy. When they remain long in the intestinal canal and the water is extracted, they become hard and may form balls or flattened masses known as scybala. These are frequently seen in convalescing typhoid patients. On the other hand, the feces may be without form, and are then liquid, either watery as in cholera or semi-fluid, pulraceous, and mushy as in diarrhea.

Odor. The odor of feces is sometimes more or less characteristic of certain conditions. Thus the stools of nursing infants have a sour smell, while in infantile diarrhea and when fermentation takes place, they have an odor of fatty acids. When urine is mixed with the stools, the odor on standing becomes ammoniacal; when blood is present, the odor is often stale. In cancerous and syphilitic and tuberculous ulcerations of the rectum the odor is so foul as to be almost overpowering.

Color. The normal color depends upon the presence of urobilin, and varies too much to be of diagnostic value. When much meat is eaten, the color is very dark. A lighter color is seen when a vegetable diet is taken. With absolute milk diet, the color almost disappears. The ingestion of some varieties of fruit, notably blackberries, and of certain drugs, as iron and bismuth, renders the stools black. Catarrh causes green stools on account of the rapid and increased discharge of unaltered bile. Unaltered bile pigments are never found in the feces normally. Santonin, rhubarb, and some cases of yellow and hematuria in red stools. In infants green stools are caused by the presence of biliverdin produced by the bacillus described by Le Sage. The feces are not so colored from the presence of unaltered blood, as the blood has undergone changes—the so-called "black" of this character. With a decrease in the amount of blood, the stools become less highly colored and if the bile is

they become clayey in color. This color is due in part to the presence of undigested fat. On the other hand if as a result of disorders of the stomach and intestine the contents pass through too rapidly, the feces may contain unaltered bile, thus giving a green or yellow color and showing the bile reaction.

Constituents.—The constituents of feces that can be recognized by the naked eye are numerous. Seeds, stones, skins of fruit and berries, and the fibers of vegetables are often seen in normal stools. In the passages of children and weak-minded individuals foreign substances of all descriptions may be present. Foreign bodies and partially digested portions of food may be mistaken for parasites. Portions of tumors from the digestive tract may appear in the feces.

In certain diseases of the stomach and small intestine, and in those who eat very fast and do not properly masticate their food, undigested and unchanged particles of food may be seen in the stools.

Gall-stones in the feces have great clinical value. Enteroliths are occasionally seen; they usually originate in the appendix.

Blood may be present in the feces in varying proportions and conditions. When found unaltered on the surface of scybalous masses, it is from the rectum or large intestine, and probably the result of traumatism. Bleeding piles may cause such an appearance, and may even cause very free hemorrhage. Severe hemorrhage may come from ulceration of the rectum or colon due to malignant disease or severe inflammation. The blood may be intimately mixed with the feces, and have its origin in the large intestine; but much more commonly its source is in the stomach or small intestine. Under such circumstances it is nearly always more or less changed by the intestinal juices, and is brownish red or black (the tarry stool mentioned above), or has the appearance of coffee-grounds. The brighter the color of the blood, the nearer is the source of the hemorrhage to the anus. The more retarded the passage, the greater the change; while if quickly expelled, blood from the small intestine may be passed unchanged, as in the hemorrhage of typhoid fever. The microscope may detect blood when the naked eye fails, but the blood corpuscles are usually destroyed unless the hemorrhage is large and quickly discharged. For blood not visible to the naked eye the occult blood tests or the spectroscope may be used. It must be remembered in connection with the latter tests that blood may be found in small amounts as a result of eating meats. It is also to be remembered that certain drugs, as already stated, may color the feces red and simulate blood.

Mucus may be present in the stools in health, but when in any marked quantity there is a catarrh of the mucous membrane of the intestines. When hard scybala are covered with mucus, or the mucus is in shreds, the large intestine is the seat of a catarrh; although it may be mixed with thin stools, as in dysentery. Usually, when the mucus is finely divided and mixed with the feces, it is from the small intestine. Mucus in large quantities and in

Parasitology of the Feces.—ANIMAL PARASITES.—A. Protozoa.—I. Rhizopoda.—This variety is made important because *Entamæba dysenteriae* belongs to it.

Entamæba dysenteriae.—*Entamæba histolytica*.—This protozoön causes amebic dysentery, an epidemic disease of tropical and subtropical countries, sporadic cases of which appear in temperate climates.

Entamæba dysenteriae vary in size from 20 to 50 μ . They are found most plentifully in the small gelatinous masses often to be seen in the feces. When not active, they are round or oblong, and highly refractive.



Entamæba dysenteriae. (Hallopeau.)

The active amebas have a characteristic movement. This consists of progression and of thrusting out and retraction of pseudopodia. Their activity varies greatly. It is best seen when the body heat is maintained. The stools should be passed into a clean warm pan and examined immediately or kept warm until examined, and a warm stage should be used with the microscope. The division into ectosarc and endosarc is usually clear during activity. The ectosarc is composed of a hyaline homogeneous mass, as are the pseudopodia, while the endosarc is made up of a dense homogeneous mass

enclosing vacuoles and a nucleus. There may be one or two large vacuoles, or the entire endosarc may appear as made up entirely of small vacuoles. The nucleus is sometimes plainly seen as a small rounded body, but is more often difficult to distinguish from the vacuoles.

The amebas will often be found to enclose bodies such as red-blood corpuscles, pus-cells, blood-coloring matter, bacilli, and micrococci.

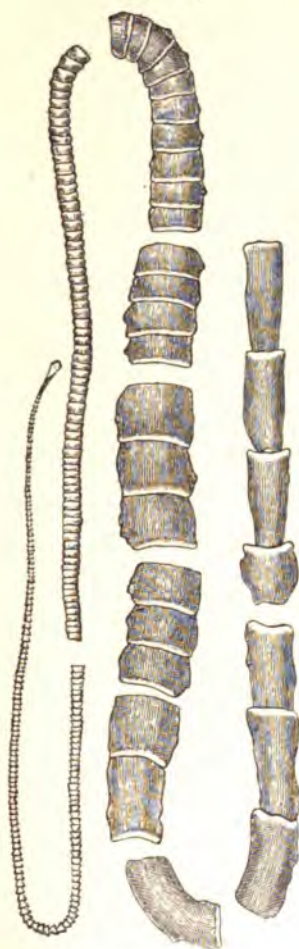
In examining feces for *Entamæba dysenteriae* it is advisable to give to the patient a saline cathartic and then examine the liquid portion of the stool, a drop of which is mounted and placed on a warm stage. If actively mobile amebas are seen, the diagnosis is assured. The *Entamæba dysenteriae* should not be confused with the *Entamæba coli*, a harmless parasite commonly found in the feces (in tropical and subtropical climates). It is a small (10 to 15 μ), grayish organism which at rest does not show the characteristic difference between the endosarc and ectosarc, and which, when motile, is sluggish in its movements.

II. *Flagellata*.—(a) *Cercomonas intestinalis* (vel *Lamblia intestinalis*, etc.) is a pyriform protozoön about 10 to 15 μ in length, nucleated, with a marked mouth-like depression on one side anteriorly, and provided with four pairs of flagella, three pairs symmetrically placed about the border of the depression mentioned and the fourth pair containing the posterior pointed extremity.

It commonly occurs in the stools of chronic diarrhea, but is of uncertain pathogenic importance.

(b) *Trichomonas intestinalis* (vel *Tr. hominis*) is about 10 or 12 μ in length, of a pyriform or subovoid shape, tapering at each extremity,

FIG. 146



Tania saginata, natural size.
(After Leuckart.)

FIG. 147



Ova of *T. solium*. a, with yolk; b, without yolk, as in mature segments. The hard brown shell is indicated. (Leuckart.)



Half-grown and mature proglottides of *Tania solium*, natural size. (After Leuckart.)

and provided with five flagella (four attached to the anterior extremity and the fifth passing from the anterior part backward as the border of an undulating membrane and extending some distance beyond the posterior extremity of the cells). It is usually encountered in cases of chronic diarrhea, but its pathogenic influence is doubtful.

III. *Infusoria*.—*Balantidium coli* (vel *Paramacium coli*) is a large ciliate infusorian of a somewhat flattened oval shape, measuring from 70 to 100 μ in its long diameter. It is colorless and uniformly covered with cilia. Its interior is finely granular, contains two contractile vacuoles, a bean-shaped nucleus, and a variable amount of extraneous substances which have been ingested from the intestinal contents. It is found in the Philippines in the stools of patients with chronic dysentery.

IV. *Sporozoa*.—*Coccidium perforans* (Leuckart) has been met in the stools of human beings occasionally, but are probably non-pathogenic.

(a) *Tænia solium* reaches a length of 2 to 3 meters. The head is the size of a pin-head. The segments forming the body are short and broad near the neck, but as they increase in size become narrower. The head appears dark, the body white. Under the microscope the head is seen to be spheroidal, with 4 pigmented sucking disks surrounding at the base a *rostellum*, which is a "crown of hooks"—chitin hooks—about 24 in number. In the ripe segments, or proglottides, is seen the longitudinal uterus with about 12 horizontal ramifications to a segment. The eggs are round or oval, 0.035 mm. long, with a thick striated shell when ripe, and contain hooklets.

FIG 158



Uncinaria duodenalis, with eggs. (After Blickhahn.)

(b) *Tænia saginata* or *mediocanellata*.—This worm is 4 to 5 meters long. The head is slightly larger than that of *T. solium* and more pigmented, and the segments are longer, thicker, and darker. The head is supplied with 4 powerful sucking cups, but has no *rostellum* or hooklets. The uterus is finely branched, and its segments have independent movement. The eggs are very similar of those of *T. solium*, but may be somewhat larger.

(c) *Tænia echinococcus*.—This tapeworm in its adult stage is a common parasite of the intestine of dogs. In its embryonic stage (cysticercus) the bladder worms are ordinarily found in the viscera or flesh of man, and a number of the mammalia. The cysticercus is peculiar in that it is capable of multiplication to the formation of large collections of the bladder worms, thus constituting the so-called "hydatid cyst." The cysticercus is a small, round or ovoid body, of a somewhat nacreous tint, varying in size from that of a buckshot to that of an ordinary Malaga grape (and even much larger), filled with a clear fluid (or in case of death of the embryo with a somewhat turbid fluid in which the hooklets of the *rostellum* are to be found) and containing the developed pyriform head of the future tapeworms, with its 4 suckers and *rostellum* of about 40 hooklets.

(d) *Hymenolepis nana* (vel *Tænia nana*).—In length, *T. nana* is 10 to 15 mm., and 0.5 mm. in breadth. The segments are all short, and at the lower end of the body are 4 times as wide as they are long. The round head, 0.3 mm. in diameter, has 4 round suckers at the base of a *rostellum* that can be inverted. At the base of the *rostellum* are about 22 hooklets. The uterus is oblong and filled with eggs. This tapeworm

has recently been found in a number of instances in this country. It is especially likely to be found in children. Its ova are nearly spherical, about 40μ in diameter, colorless, with double wall. The embryo within measures about 22 to 23μ , is colorless and granular, and is provided with six embryonic hooklets, easily discernible.

(e) *Hymenolepis flavopunctata* (vel *Tania flavopunctata*).—This small tapeworm has been encountered several times in the United States. The head is small, clubbed, and unarmed. Each fully developed link is marked by a small yellow spot in the posterior portion. The ova are similar in appearance to those of *H. nana*, but larger, measuring 60μ in diameter.

(f) *Dibothriocephalus latus*.—This is the largest of the tapeworms likely to be met in the human intestine, measuring 7 or 8 meters or more in length. The head is of an elongated club shape, provided with two long, narrow slits one on each side, serving as suckers, and without hooklets or rostellum. The proglottides are short near the head and very wide, but toward the lower end of the worm become nearly square. Each ripe link shows a reddish or brownish rosette-like mass near the middle, caused by the coiled uterine tube filled with the yellowish or faintly brownish eggs. These eggs may be encountered in the dejecta, as oval, thin-shelled, brownish objects measuring about 70μ in length and 40 to 45μ in breadth, showing more or less advanced segmentation and provided with an operculum or lid at one end for emergence of the ciliated embryo. The embryo makes its habitat in the tissues of certain fish.

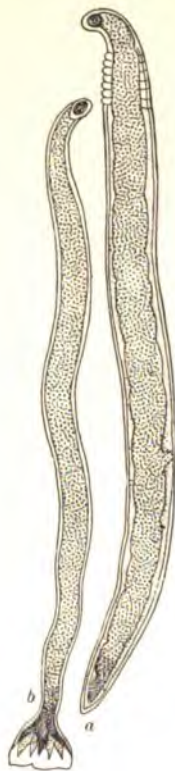
It will not be necessary to describe certain other varieties that are rarely met with.

II. *Trematodes* (Flukeworms).—(a) *Fasciola hepatica* (*Distomum hepaticum*) measures 28 mm. by 10 mm., and is shaped like a leaf. A short head is situated at the broad end and has one sucker; on the under surface is another sucker, and between the two is the opening of the uterus, a highly convoluted arrangement. This worm is not often seen in man. Its habitat is the hepatic ducts of sheep, cattle, swine, etc. The worm is hermaphroditic, and its eggs after discharge from the genital pore, find their way along the bile ducts and into the intestine. They are thin-walled, of a brownish-yellow hue, ovoid in shape, with a lid on one end for the emergence of the embryo, usually seen in advanced segmentation and measure from 120 to 140μ in length and 70 to 90μ in width.

(b) *Distoma* (*Dicrocoelium*) *lanceolatum*.—This fluke preserves the general leaf-shape of the *Distomum hepaticum*. It is about 8 mm. long and 1.5 to 2.5 mm. broad. The extremities, especially the anterior, are sharper than those of the preceding. It is semitransparent, and flecked with brown from the eggs within the body. It is more common in the lower animals than in man. Its habitat is in the bile passages, its eggs being carried thence into the intestine. They are ovoid, with thin shell, of a light brown color, measuring about 40 to 45μ in length and 20 to

B. *Platyhelminthes*.—Vermineous intestinal parasites are more generally known than the above protozoa, and their clinical importance less uncertain. Both local and general symptoms of importance may be predicated of individual examples.

FIG. 148



Uncinaria duodenalis, magnified. a, female; b, male. (Bristowe.)

FIG. 149



Head of *Tænia saginata*. (Eichhorst.)

FIG. 150

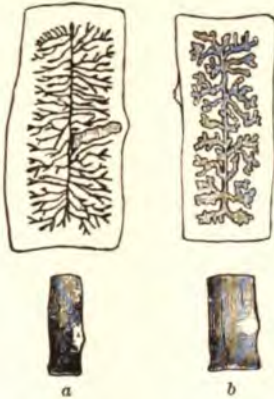


Head of *T. solium*. $\times 45$.

I. *Cestodes*.—*Tapeworms*.—These parasites infest only intestine, to the walls of which they cling by the head. The neck are small; the joints are flat and form long ribbons. joints continually drop off and can easily be recognized in by the naked eye, and the eggs by the use of the microsc

feces are best washed in water and broken up to obtain the eggs. As the lower joints are lost, new ones take their place from above. The more important are the following:

FIG. 151



Proglottides of (a) *Tania saginata* and (b) *T. solium*, natural size, and enlarged three times to show arrangement of uterus. (After Leuckart.)

FIG. 152



Proglottis of *Bothriocephalus latus*, natural size, and enlarged three times. (After Eichhorst.)

FIG. 153



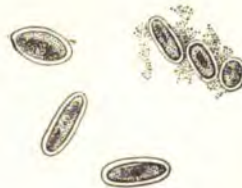
Eggs of (a) *T. saginata*; (b) *T. solium*; (c) *Bothriocephalus latus*. $\times 300$. (After Eichhorst.)

FIG. 154



Eggs of *Tricocephalus dispar*. $\times 275$. (Eichhorst.)

FIG. 155



Eggs of *Oxyuris vermicularis*. $\times 275$. (After Eichhorst.)

FIG. 156



Oxyuris vermicularis, natural size. (Eichhorst.)

FIG. 157



Tricocephalus dispar, male and female, natural size. (Eichhorst.)

ditions these develop into sexually mature rhabditoid males and females (*Anguillula stercoralis*), and the females deposit ova or living young, very like those produced in the intestine but a little more slender, which gain access to the digestive tract of another host, where they pass their parasitic life as *Anguillula intestinalis*. The parasite is the cause of Cochin-China diarrhea, and is found in the stools of cases of endemic diarrhea of hot countries.

VEGETABLE PARASITES.—We find both *pathogenic* and *non-pathogenic* vegetable parasites in the feces, which are classed as (1) moulds, (2) yeasts, and (3) fission-fungi.

1. *Moulds.*—The thrush fungus is of rare occurrence in the feces, and has no special clinical import.

2. *Yeasts.*—In all feces, yeast cells exist. They are most numerous in acid stools. They stain dark brown with a solution of iodine and potassium iodide.

3. *Fission-fungi.*—Bacteria are found in great numbers and make up from 20 to 40 per cent. of the dried stool. Their differentiation and recognition requires special bacteriological studies, excepting tubercle bacilli, which can be stained and recognized as in sputum. They are found occasionally in pulmonary tuberculosis (swallowed sputum), but the constant recovery of them in large numbers is indicative of intestinal involvement.

Chemical Examination of the Feces.—The chemical examination of the feces has but slight clinical value.

The normal freshly passed feces are usually alkaline but the reaction is not constant. An excessive fat or carbohydrate diet may cause them to become acid, and in intestinal diseases they may be acid or alkali.

Mucin and albumin are normally present; peptones may occur in different diseases. (Von Jaksch.) Among the acids to be found are bile acids, volatile and fatty acids, formic, acetic, butyric, and propionic acids; while phenol, indol, skatol, cholesterin, and fats, according to von Jaksch, are always present. The demonstration of these substances will not aid in the diagnosis.

The normal *coloring matter* of the feces is stercobilin (urobilin) and pigments derived from the food. The presence of urobilin may be shown by treating a small portion of the feces with a saturated aqueous solution of bichloride of mercury. Normal feces are colored red; a green coloration shows the presence of unaltered bile pigments.

Test of Occult Blood.—The most important chemical examinations for ordinary purposes are the tests for occult blood. The patient should be placed on a meat-free diet for three days preceding the examination, and all local bleeding-points, as in the nose, pharynx, lungs, rectum, etc., must be excluded.

Alain Test.—Grind up a small piece of feces in 5 c.c. of water and place in a test-tube, add ether and shake well and pour off supernatant fluid. Repeat twice in order to thoroughly extract fats. Add one-third volume of glacial acetic acid and equal volume of ether. Shake

well and pour off about 3 c.c. of ethereal layer. To this add about 3 c.c. of a solution of aloin (sufficient to cover the end of a spatula) in 70 per cent. alcohol. The solution should be freshly made. Add about 3 c.c. of ozonized turpentine (c. p. turpentine exposed to the air for a month). In the presence of blood the lower solution in the test-tube, the aloin solution, turns a cherry red within fifteen minutes. If the test is negative the yellow solution remains uncolored for fifteen minutes, but after this time may begin to turn red.

Benzidin Test.—Boil a small portion of the feces in a test-tube. Cool. Add 2 to 3 c.c. of a concentrated solution of benzidin (sufficient to cover the end of a knife blade) and glacial acetic acid (5 c.c.). Add about 3 c.c. of hydrogen peroxide (3 per cent.) and shake. The white foam will contain bluish particles and in two or three minutes the entire solution will be colored dark blue or green blue, which gradually becomes so intense as to be almost black. This test is extremely delicate, reacting to blood diluted 1 to 100,000.

Value of Tests.—These tests are of great value in diagnosing disorders of the alimentary canal. If bleeding elsewhere (gums, lungs, rectum, etc.) is excluded and the patient has been on a meat-free diet the presence of occult blood is highly suggestive of malignant or ulcerative disease of the gastro-intestinal tract. In cancer of the stomach, the test is positive early and continuously; in ulcer of the stomach, when subacute or acute it is positive; in chronic ulcer it is intermittently positive. Neoplastic ulcerations of the intestines are positive; other ulcerations vary; intestinal tuberculosis is usually negative.

CHAPTER XXIX

EXPLORATORY PUNCTURE

THE presence or absence of fluids in the natural cavities of the body, as the pericardium, the pleura, the abdomen, or the gall-bladder, must frequently be ascertained by means of puncture or aspiration. The fluid is secured for examination at the same time. The fluid of tumors or cysts is likewise withdrawn to complete a diagnosis by determining its chemical, microscopic, or bacteriological character.

The Instruments.—If it is the desire of the observer to determine the presence of fluid, or if fluid is to be obtained for examination, a syringe or aspirator must be used. An ordinary antitoxin syringe may be used in most cases. A special aspirator made for diagnosis by instrument makers is better. The needles are sufficiently long; the barrel large enough to hold sufficient fluid for any method of examination. If the diagnosis is to be followed by aspiration, the apparatus of Dieulafoy, or any equally perfect apparatus, may be used at once. An exploring trocar is used for the removal of pieces of solid tissue, as from muscle or from tumors of the lung.

Preparation of Instrument and Skin.—The instruments should be sterilized in a steam sterilizer or boiled, and carried to the patient without contamination. The overlying skin should be thoroughly scrubbed and covered with tincture of iodine a short time before, and immediately before the puncture. At the time of puncture the surface should be anesthetized by the ethyl chloride spray, or by subcutaneous anesthesia with cocaine.

The Point of Puncture.—The points selected for aspiration depend upon the cavity to be explored or the situation of the cyst.

The Pleura.—To withdraw fluid within the pleura, it is best to select a point for aspiration in one of the lower interspaces of the chest, because the fluid is more likely to accumulate in this position and because complete aspiration can there be performed if necessary. The sixth or seventh interspace in the anterior axillary line, or the eighth or ninth interspace in the posterior axillary or scapular line, may be selected. On the right side the upper interspace of the two should be chosen on account of the position of the liver. If the contents tend to point or break out at any particular spot on the surface of the chest, the puncture may be made in this area. In suspected loculated empyema or effusions, the point of puncture should be at the site of greatest dulness and least fremitus.

The Pericardium.—For aspiration of the pericardium three points of election have been recommended: first, the usual position of the apex-

beat, in the fifth interspace, inside of the midclavicular line; second, the space between the ensiform cartilage and the left seventh cartilage; third, Rotch has tapped the fifth right interspace a number of times on the cadaver, and thinks that this situation is a proper one in the living subject. Care must be taken to insert the needle slowly and with the point directed downward and toward the left axilla when this position is selected.

FIG. 159



Aspiration of a pleural effusion.

The Abdomen.—Explorations of this character are probably more feasible in connection with diseases of the *liver*. It does not appear to be harmful to insert needles into that organ, and valuable information is often gained thereby.

The median line should be selected for the puncture in aspiration of the *abdomen*, to determine the character of the peritoneal contents. The bladder must be emptied and a point midway between the umbilicus and pubes selected.

The Vertebral Canal.—**Spinal or Lumbar Puncture.**—The puncture is made with a syringe or a fine trocar and cannula. The syringe itself may be removed and the fluid allowed to ooze through the needle drop by drop. A needle 4 cm. in length and 1 mm. in

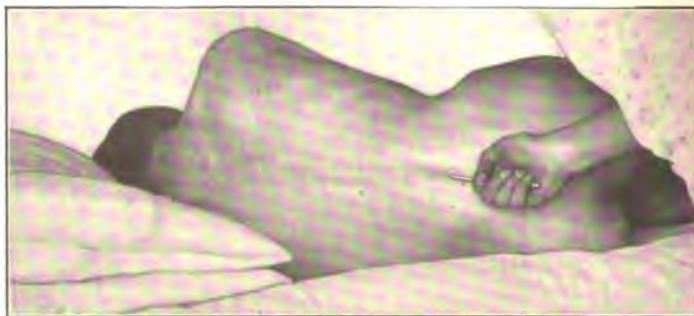
diameter is suitable for infants, a longer needle for children over ten years and for adults. Ordinarily the puncture is made between the third and fourth lumbar vertebræ to one side or the other of the median line. The point at which the needle enters the skin should be the level of the lower end of the spinous process of the fourth lumbar vertebra, one-half inch to one side. The position of the

FIG. 160



Lumbar puncture.

FIG. 161



Lumbar puncture—introducing the trocar.

fourth lumbar vertebra may be determined in one of three ways: (1) by counting from the *vertebra prominens*; (2) the method of Jacoby which depends upon the fact that a line drawn through the crests of the ilia across the back intersects the spinous process of the fourth lumbar vertebra; (3) the spinous process of the fifth lumbar vertebra is considerably more prominent than the spinous processes

of the sacrum, and this peculiarity may be utilized as a check upon the other two methods. Practically in all cases all three methods should be employed. A mark on the skin may be made with an indelible pencil or with a soft lead pencil.

Technique.—The technique is as follows: the patient may be either sitting up or lying on one side. The former position can only be used in patients who are conscious and not suffering from severe disease. The patient sits preferably crosswise upon a narrow operating table; the body is bent well forward, separating the spines of the vertebræ as much as possible. The latter position should be used if the patient is seriously ill. The patient should lie upon the side with the thighs flexed upon the abdomen and the thorax bent over the knees. The site of the puncture should be surrounded by sterile towels. The point of the needle is placed directly over the mark upon the skin; the needle is held at an angle of 45 degrees to the surface of the back, and inclined slightly toward the median line. It should be thrust forward steadily and slowly, and not turned or twisted. In the great majority of instances, if the patient's back is well arched and the needle is thrust in the direction described, it will enter the spinal canal without difficulty. When it has been inserted between 6 and 9 cm., if it has not met with obstruction, it can be assumed that it is in the lumen of the canal and the trocar may be withdrawn. As soon as the trocar has been withdrawn, ordinarily two or three drops of clear or bloody fluid will flow from the tube. A mercury manometer at the level of the puncture should be connected by a rubber tube with the cannula and the intraspinal pressure determined. The fluid can then be permitted to flow into prepared receptacles until it begins to drop very slowly; the needle should then be withdrawn and the opening sealed with a little cotton and collodion.

Certain *modifications* may be employed. A preliminary incision sufficiently large to admit the needle without touching the skin on either side may be made at the site of the puncture to prevent carrying in skin bacteria, although infection practically never follows a simple puncture. The puncture may be made in any of the other interlumbar spaces where the difficulties are not much greater.

Of the *accidents* that may happen during the operation, the most important is the faulty direction of the needle, so that instead of finding the intervertebral space it strikes upon the arch of the lumbar vertebra. If this occurs, it is only necessary to withdraw the needle a short distance, alter its direction slightly, and thrust again. In some individuals the interspaces are so close together that it is impossible to insert a needle of ordinary size, and the operation becomes futile. If the cerebrospinal pressure is low, and the needle is very dull, it may be difficult to pierce the spinal membranes and no fluid is obtained. If the needle, in piercing the canal, wounds one of the large veins, the spinal fluid will be bloody, and this may render its subsequent examination unsatisfactory. If it presses against one of the roots of the cauda

equina, it may cause pain or twitching of the muscles of the extremities. Sometimes pseudomembrane with pus or gelatinous fluid fills the spinal canal, and hence no liquid is withdrawn. Adhesions may cause the fluid to be loculated.

Sometimes when the needle has been inserted and the spinal fluid has flowed freely, it may suddenly stop. This is due to some obstruction, usually a nerve root, pressing against the end of the needle. It can usually be dislodged by reinserting the trocar. The cerebrospinal fluid should flow out rather slowly, preferably drop by drop. If it comes in a forcible stream, its flow should be checked either by pinching the rubber tube, or, if sitting up, having the patient lie down. If during the operation the patient should complain of headache, dizziness, or nausea, or if he groans, the operation should be interrupted at once and the patient placed either horizontally or with the lower portion of the body slightly elevated.

Data Obtained from Spinal Puncture.—The normal pressure of the cerebrospinal fluid when the puncture is made below the *conus terminalis* and the patient is upright is between 40 and 100 mm. If the patient is lying down, the pressure ranges between 0 and 40 mm. If the pressure is above 150 mm., with the patient in the upright position, it is distinctly pathological. In some cases it may even reach 800 mm. Increase in the cerebrospinal pressure indicates irritation of the central nervous system, due to the presence of a tumor, or to some toxic substance in the blood, as for example in uremia and lead-poisoning, or to some inflammatory process in the cerebrospinal system, particularly meningitis.

The Fluid.—This is clear normally and in non-inflammatory cerebral and spinal meningeal disorders and in tuberculous meningitis; in other forms of meningitis it is cloudy and turbid. Blood may be found in lateral ventricle hemorrhage.

Chemical Examination of the Cerebrospinal Fluid.—Ordinarily the cerebrospinal fluid contains a mere trace of albumin. Under pathological conditions, it may contain as much as 5 to 10 per cent., particularly in tuberculous meningitis. If the fluid is bloody, the percentage of albumin may be considerably increased without indicating a pathological change. It contains a substance—pyrocatechin—that reduces Fehling's solution. This ordinarily disappears a few hours after the fluid has been withdrawn.

Noguchi's Butyric Acid Test.—The test is made to determine the globulin content of the cerebrospinal fluid (or blood-serum), which is markedly increased in syphilitic and parasymphilitic disorders. Take 0.2 c.c. of the fluid, free from blood; add 0.3 c.c. of butyric acid in normal saline; boil five minutes; add 0.1 c.c. normal sodium hydroxide solution and boil for thirty seconds. Increased globulin content is shown by the presence of a granular or flocculent precipitate, appearing within two hours. The test is also frequently positive in tuberculous meningitis and occasionally in other forms of meningitis.

The Wassermann reaction may also be applied to the cerebrospinal fluid. The fluid may be searched for tubercle bacilli in suspected cases of tuberculous meningitis. The presence of large numbers of mononuclear leukocytes would exclude the other forms of meningitis.

Cytological Examination.—The fluid should be centrifugated, and the sediment spread upon cover-glasses and stained by the usual methods for blood. Normally there are but few cells (lymphocytes) or none at all. In meningeal disease, if the polymorphonuclear forms are in excess, acute meningitis is probably present; if the mononuclear forms of leukocytes predominate, the infection is presumably tuberculous.

In syphilitic and parasyphilitic disorders (tabes, paresis, cerebrospinal syphilis) there is also a marked lymphocytic increase.

Bacteriological Examination.—Cover-glass preparations are made of the fluid, and cultures taken at once. Streptococci, staphylococci, the pneumococcus, and the meningococcus (*Diplococcus intracellularis*) may be detected in purulent meningitis. Tubercle bacilli have been found in tuberculous meningitis, especially after centrifugation or sedimentation. After the fluid has been twenty-four hours in a conical glass the fine clots which form should be examined for bacilli. The absence of bacilli does not exclude tuberculosis. The positive result, however, is diagnostic. Inoculations of guinea-pigs will give a positive diagnosis.

Cysts and Tumors.—These with fluid contents, should be punctured over the point which presents externally, and where they are evidently in closest proximity to the external walls.

The Spleen.—The spleen has been punctured for therapeutic and diagnostic purposes. If the organ is hard, as in chronic malaria, it may be done without danger; but if it is enlarged and soft, as in infectious diseases, such as typhoid fever, it is hardly justifiable to puncture it, because of the danger of subsequent rupture. Risk attends the puncture of other organs, as the kidney.

THE EXAMINATION OF EXUDATES AND TRANSUDATES

The serous cavities contain but little fluid; pathological excess of fluid may be inflammatory in origin, an *exudate*, or due to non-inflammatory circulatory disorders, a *transudate*.

Fluids are also obtained from cysts, but do not require different methods of examination. In any case the fluid is withdrawn by exploratory puncture. In the examination of the fluid the naked-eye appearances are first noted; then microscopic examination with and without staining is resorted to. Chemical examination is also required. Often culture preparations and inoculations must be resorted to, as in the case of pus or of serous exudation.

The Exudates.—They may be composed of pus, seropus, gangrenous debris, blood, or pure serum. When pus, seropus, or putrid fluid is

withdrawn, it implies absolutely an inflammatory origin. Blood or bloody serum is usually of tuberculous or cancerous origin. A chylous fluid is usually due to obstruction of the lymph channels.

PURULENT EXUDATES.—Pus ranges in color from gray to greenish yellow. It is turbid, of high specific gravity, and alkaline. It varies in consistence. When standing after removal it separates into two layers: the upper layer is light yellow and transparent, and the lower opaque. Pus may be mixed with blood, and is then reddish brown. When it has undergone decomposition, it is thin, green, or brownish red, and of a penetrating odor.

Microscopic Examination.—If fresh the specimen contains innumerable leukocytes of the polynuclear neutrophilic variety, which may show ameboid movements. If old, the leukocytes are degenerated and the nuclei are made out with difficulty, if at all, and the granules cannot be seen. Red-blood cells are usually also found. Their appearance depends upon the freshness of the hemorrhage. Hematoidin (if there has been previous hemorrhage), fatty acid crystals and triple phosphates are seen in pus and cholesterol crystals in addition in old pus. Bacteria are also found, but their proper identification depends upon cultural or inoculation methods.

SEMPURULENT EXUDATES.—They resemble purulent discharges, chemically and morphologically. They point to antecedent inflammation.

PETRIE EXUDATES.—The exudates are brown or brownish green in color. The odor is penetrating and offensive. They are usually alkaline in reaction. On *microscopic examination* old leukocytes and crystals of fat, cholesterol and hematoidin are seen; fission-fungi of various forms are also seen.

HEMORRHAGIC EXUDATES.—Hemorrhagic exudates contain red-blood corpuscles and hemoglobin in large amount. Fatty epithelial cells are found. Quinke states that when the glycogen reaction is shown, carcinoma is probably present. Hemorrhagic exudates are due most frequently to cancer, to tubercle, or to scurvy.

STRAWS EXUDATES.—The fluid is clear and light yellow or straw-colored. On standing a white fibrous clot is deposited. On *microscopic examination* red-blood corpuscles, leukocytes, fatty globules, and epithelial cells are found. They may be bunched in groups or scattered about. The microorganisms, if present, are detected with difficulty. Cholesterol crystals are found in old serum. On *chemical examination* the fluid contains more than 5 per cent. of serum albumin and globulin, peptone is absent in purulent exudates, sugar in small amounts and acetic acid is found. The specific gravity of the fluid is above 1.015.

CHYLOUS EXUDATES.—This style is found in fluids of low specific gravity. Such an effusion is not in the and is due to leakage of lymphatics into the peritoneal cavity. It is known as a chylous effusion. Chylous effusion is a term applied to the second variety of effusion

mentioned in this section. The fluid has the property of chyle. Sometimes in peritoneal exudation, particularly if the patient has been upon a milk diet, the fluid contains fatty matter, which gives it a milky appearance. The same character of fluid is seen in obstruction of the thoracic duct.

Transudates.—This class of fluids is serous, bloody, or chylous. The specific gravity is lower than in inflammatory effusion. The color is light and the reaction usually alkaline. On microscopic examination but little is found. In pleuritic effusions there may be considerable endothelium, which, if mixed with blood, may be due to carcinoma. Serum contains albumin and sugar, the former in great excess. Peptone is always absent. The fluid coagulates with difficulty on boiling.

Differentiation between Exudates and Transudates.—This is obtained by comparison of the specific gravity and albumin content of the two fluids. The transudation that occurs in cachectic and obstructive conditions has a specific gravity of 1.005 to 1.015 and the albumin rarely is greater than 2.5 per cent.; the exudation of inflammatory conditions has a specific gravity greater than 1.018 and the albumin content is between 4 and 6 per cent. Furthermore, a transudate upon microscopic examination shows only a few cells and a preponderance of endothelial cells as a rule; an exudate shows numerous cells, the leukocytes predominating.

Cytodiagnosis.—The microscopic study of the leukocytes of an exudate yields valuable information, particularly in regard to the nature of pleuritic effusions and to a somewhat lesser extent in other effusions. A very thin smear is made, dried rapidly, and stained with eosin and methylene blue. Acute inflammatory conditions show a marked predominance of the polynuclear neutrophiles (averaging 70 per cent.); chronic inflammatory conditions, as tuberculosis, show a predominance of the lymphocytes (50 to 95 per cent.), the percentage increasing with the length of time the effusion is present. The transudate of non-inflammatory circulatory disorders also shows a high lymphocytosis, but is differentiated from tuberculous effusion by the usual methods of differentiating between an exudate and a transudate.

In malignant disease of the serous membranes the occurrence of mitotic endothelial cells is highly suggestive. If pieces of tissue are removed, the diagnosis can be verified by histological examination of the tissue.

Contents of Cysts.—In aspiration of the abdomen and of the pleura, cysts are sometimes evacuated, the nature of which is often determined by an examination of the fluid.

Hydatid Cysts.—The fluid of hydatid cysts is clear, alkaline, and of a specific gravity of 1.010. It contains sodium chloride in excess, grape sugar in small amount, and very little if any albumin. On microscopic examination hooklets are found, as in the sputum from hydatid cyst of the lung, as well as portions of membrane. The heads or scolices are sometimes found. If suppuration has taken place, the original

nature of the cyst cannot be made out unless hooklets are found. After the fluid has been standing in a conical glass vessel the bodies may be found in the sediment.

Ovarian Cysts.—The fluid from an ovarian cyst is of high specific gravity, averaging 1.022, of alkaline reaction, and contains usually but a small amount of albumin. On microscopic examination various forms of epithelial cells are seen, colloid bodies, and cholesterin crystals. If hemorrhage has taken place in the cyst, the color of the fluid is correspondingly changed, and besides the squamous, columnar, and ciliated varieties, some epithelium in the stage of fatty degeneration and red and white blood corpuscles are seen. In colloid cysts the usual concretions are found.

In *dermoid* cysts, in addition to the above, squamous epithelium, hairs, and fatty hematoidin and cholesterin crystals are detected. *Ovarian fluid* contains albumin and pseudomucin (metalbumin). The latter is detected by mixing a portion of the fluid with three times its bulk of alcohol. It is then allowed to stand for twenty-four hours, when it is filtered. The precipitate is removed and suspended in water. After filtering, the filtrate is seen to be opalescent, and is tested as follows:

1. On boiling no precipitate is formed, but the fluid becomes turbid.
2. The fluid becomes thick and of a yellowish tint when treated with acetic acid alone.

Cystic Kidney.—The fluid from a cystic kidney can be recognized by the properties it derives from the renal secretion. Urea and uric acid in large amounts point to its true source. Renal epithelium is of the greatest diagnostic value. (See Urine.)

Pancreatic Cysts.—The fluid from cysts of the pancreas is of a specific gravity of 1.012, but may be as high as 1.028. It contains cholesterin crystals in abundance, and blood and pigment. Serum-albumin is present, but metalbumin is not found. Of particular diagnostic value is the fact that the fluid digests fibrin and albumin and emulsifies fat. Less important is the conversion of starch into sugar, as other cystic fluids have the same property.

CHAPTER XXX

FUNCTIONAL TESTS OF ORGANIC EFFICIENCY

THE study of the functions of organs supposedly pathological, has in the past few years received much attention, and as a result of this work much knowledge has been gained as to the capacity of an organ for performing the work it is called upon to do. Valuable information is gathered by these studies, and such information is found to be of importance not only in diagnosis but also in treatment and in prognosis. Broadly speaking, the study of the pathological physiology of the organs, includes most of our present-day methods of diagnosis. As for example, the study of the motor and secretory power of the stomach by means of the stomach-tube, the examination of the blood or of the stools, and the various examinations for impairment of the functions of the cerebrospinal system, all aid in determining functional capacity; but in its more limited sense, the term "functional test" is applied to the specific method employed (1) in the diagnosis of disorders of organs inaccessible to the ordinary methods of diagnosis, by means of the study of their functions and (2) in the more accurate and early diagnosis of the extent of impairment of the functions of an organ, the more apparent disorders of which can be appreciated by the common diagnostic methods.

The importance of functional tests cannot be gainsaid; not only in diagnosis but in treatment have they proved of paramount value. In treatment, we are concerned not so much with what the disease is, as we are with the predominating disturbance of function the disease produces. In the prognosis of a case we are aided in foretelling the results by the degree of impairment in function the disease produces.

In the present section four organs will be considered, the pancreas and the liver, organs which for the most part, unless grossly diseased, are incapable of causing definite manifestations of their disorders, and the heart and kidneys, organs which cause definite manifestations when diseased, but in which we are unable to appreciate by ordinary means the extent of the damage and the effects of the disorder even when great impairment of the function of that organ has taken place.

Other organs have been omitted either because no satisfactory clinical tests of functional efficiency have been devised, as with the lungs and with cancer of the stomach, or because the usual methods of diagnosis give sufficient information as with the stomach and intestines, or because the tests that have been suggested are too unsafe, as in giving thyroid extract to cases of thyroid disease and observing the results.

Liver.—A good functional test depends largely upon the possibility of testing for impairment of the function that is peculiar to that organ. The liver is an extremely complex organ and most of its functions are

performed in collaboration with other organs and so depend upon numerous extraneous factors. Furthermore the more obvious functions of the liver are known to take place even when there is manifestly advanced disease of the organ. It follows, then, that the tests for liver sufficiency must depend upon so many factors that they strongly mitigate against specific tests. Such is actually the case. No functional tests have yet been devised that will specifically denote insufficiency of the hepatic parenchyma. The following tests are more or less valuable as adjuncts to other findings, particularly when repeated several times with the same results.

1. **Alimentary Levulosuria (Strauss Test).**—100 grams of levulose dissolved in weak tea or hot water are given on an empty stomach. The urine is then collected every hour for four hours. The several specimens are then tested for a reducing substance by the usual method of examining for sugar. The presence of alimentary levulosuria is supposed to indicate insufficiency of the liver. In 86.9 per cent. of cases of cirrhosis of the liver the test was positive (Chajes). However, the test is frequently positive in certain percentages of apparently healthy individuals. Hence, the test should be repeated and found positive at least twice before considering the result as even suggestive.

2. **Urobilinuria.**—Based upon the hypothesis that pathological urobilin originates in the diseased liver the presence of a pronounced urobilinuria is supposedly indicative of hepatic insufficiency. As the hypothesis, however, is considered by many erroneous, the specificity of the test is very questionable. Be that as it may, the empirical fact remains that urobilin is extremely common in hepatic cirrhosis, in passive congestion of the liver and in febrile conditions, probably from an associated cloudy swelling of the parenchyma. It is found frequently in pernicious anemia, carcinoma, extra-uterine pregnancy, secondary syphilis, hemophilia, etc., hence the test is only a confirmatory finding in association with other findings. The test for urobilinuria (Gerhardt's test) is performed as follows: 10 c.c. of urine are extracted with chloroform by shaking. Add 5 drops of Lugol's solution (liq. iodi. comp. U. S. P.) to the chloroform extract and follow with a dilute solution of sodium hydrate. The extract is colored a yellowish brown and shows a superb greenish fluorescence when urobilin is present.

Pancreas.—This organ is inaccessible to ordinary methods of examination and the diagnosis of pancreatic disease depends entirely upon examinations showing alterations in the function of the organ. The various tests are applicable only in chronic pancreatic disease in which valuable diagnostic data is also achieved by the examination of the stools after a test diet.¹ The stools are found to be bulky, much increased

¹ Schmidt's Test Diet. For three days before the examination the patient should take daily the following diet: milk, 1.5 liters; biscuits, 100 grams; oatmeal, 80 grams; 2 eggs; butter, 50 grams; minced beef, 125 grams; potatoes, 300 grams. The beef is lightly fried in butter so that the interior is rare. The oatmeal is prepared as a gruel with some of the milk and the butter, and the potatoes are prepared as a purée. Milk and biscuits are taken in the morning and afternoon, oatmeal gruel and the eggs in the forenoon and evening, and the remainder of the meal at mid-day.

in weight when dried (normal 54 grams, increase up to 438 grams), fatty, light in color, with hydrobilirubin present (excluding the stools of jaundice), and showing microscopically many fat droplets and crystals and flakes of fatty acids and soap, and, most important, large numbers of yellow muscle fibers with well-preserved striations (creatorrhea).

The specific tests for pancreatic insufficiency include many tests which have for their purpose the estimation of the activity of the pancreatic enzymes. *Schmidt's cell-nuclei* test consists of the ingestion of pieces of thymus 0.5 cm. in size hardened in alcohol and enclosed in gauze bags which are recovered in the stools. Preservation of the nuclei (which must be stained) indicates pancreatic insufficiency.

By means of Einhorn's duodenal tube, a thin rubber tube with a small multiperforated metallic acorn-shaped bulb on the end, the duodenal contents can be recovered and examined for trypsin. The metal end of the tube is swallowed and left in the stomach for two to four hours, at the end of which time it has presumably passed into the duodenum. A strong syringe is attached to the end projecting from the mouth and the contents aspirated from the duodenum. If the bulb is in the duodenum a golden-yellow fluid is recovered. This is tested for trypsin by placing a measured quantity of casein (2 per cent.) in a test-tube, adding ascending amounts of duodenal contents and incubating for forty-five minutes, or placing in a water-bath for the same length of time. The presence of casein is then tested for by adding acetic acid and alcohol solution (ãã 25 per cent.). Cloudiness of the solution occurs if the casein is not entirely digested. The trypsin estimation is made in percentage of trypsin required to digest the casein.

The presence of trypsin may also be sought for in the stomach contents by giving a test-meal of 200 c.c. of olive oil and removing by lavage in forty-five minutes. Trypsin is also tested for in the stools by means of casein digestion. Absence of digestion of casein in any of these tests indicates pancreatic insufficiency. Diastase in the feces is also estimated quantitatively as a test for pancreatic sufficiency.

In the study of disturbances of the internal secretions of the pancreas the alimentary glycosuria test is employed (see Diabetes). As with the various tests for hepatic sufficiency none of the above tests can be said to be pathognomonic, but the occurrence of several of the tests in association with other findings is extremely suggestive. Cammidge's test, at one time hailed as a positive test of pancreatic disease, has been shown to be valueless. It may be positive in health and in many diseases in which the pancreas is normal, while it is often negative when the pancreas is diseased.

Kidney.—The recent tests for kidney sufficiency have given splendid results and have been shown to be of considerable value in diagnosis and treatment. Various tests have been proposed, but for ordinary clinical purposes the indiocarmine or phenosulphonaphthalein tests are the most satisfactory, and of these two the latter is probably the better. This test, devised by Rowntree and Geraghty, is performed as follows:

The patient drinks 300 to 400 c.c. of water twenty minutes before the test. Exactly 1 c.c. of the standard solution of phthalein, containing 0 mg. of the drug to the centimeter of salt solution, is injected into the muscle immediately after the patient has emptied the bladder or has been catheterized. The time of first appearance of the dye is estimated by having the patient void every five minutes until its appearance as indicated by a red tint to the urine upon addition of sodium hydroxide. Normally this should take place within ten minutes. Cases of delayed phthalein elimination are usually found to have a low total output. (In cases of urinary obstruction, the bladder should be emptied with a catheter which is left *in situ*. The first appearance of the dye is noted by having the catheter drain into a flask containing the alkali). The patient then voids at the end of an hour and again at the end of the second hour, or the urine passed through the catheter is collected at the end of an hour and an hour after this first hour and the amount of drug excreted estimated, preferably by a Dubosq colorimeter or the Autocolor-Königsberger colorimeter as modified by Rowntree and Goughay. Sufficiently accurate results are obtained by means of the Dunning colorimeter,¹ in which the urine is compared with cylinders containing different percentages of the dye prepared in such a way as to be kept permanently. The urine, no matter which colorimeter is used, is filtered and rendered distinctly alkaline by the addition of a 20 per cent. solution of hydroxide. Sufficient distilled water is then added to make the solution measure one liter. If the color is very faint add only enough water to bring solution up to 300 c.c. and multiply the result by 2. With the Dunning colorimeter an open ampoule is filled with the solution and placed in one of the compartments of a box with two compartments. In the other compartment one of the differently colored sealed ampoules is placed until one is found which accurately matches the solution in color. The percentage is indicated by a figure on the test ampoule. With the other colorimeters a standard alkaline solution containing 1 mg. of phthalein to the liter is placed in one of the cups and the urine solution in the other. The test-cup is then manipulated until the two sides of the prism held as seen through the lenses accurately match each other. The percentage is directly read from the indicator wheel on the manipulating screw.

Normally there is elimination of from 40 to 60 per cent. of the phthalein in the first hour and from 30 to 50 per cent. in the second hour. Absent or diminished elimination of the dye is an indication of renal insufficiency; the degree is indicated by the amount of dye eliminated. The test is of value in diagnosis of following: the degree of impairment of the kidney function; a differentiating degree of condition from conditions simulating it; and the condition of the kidney showing its action after the kidney has been exposed to the various conditions. It is most useful in cases

¹ Dunning, J. *Journal of the American Medical Association*, 1916, 61, 1000.

the results achieved by the therapeutic measures, and when operative measures are to be instituted, it indicates the time the operation should be performed, that is, after preliminary treatment it will show when the kidney is acting sufficiently well to operate, and by means of ureteral catheterization the test will show the amount of work done by each kidney if nephrectomy for any cause is indicated. In prognosis, impairment in the amount of dye eliminated shows impairment in the kidney function. With elimination of less than 10 per cent. of the phthalein in two hours the prognosis is extremely grave and the patient usually dies in a short time.

The proved worth of this test and the simplicity of its technique renders useless the need of describing other less valuable tests.

Recently, Schlayer has been experimenting with the purpose of devising tests to locate the anatomical lesions in nephritis. He injects intravenously 10 gm. of milk-sugar in solution. If elimination does not occur within six hours, the glomeruli are involved. This work has been confirmed but the methods he devised to presumably show tubular involvement have apparently been shown to be of but little value.

Heart.—The estimation of the functional capacity of the heart is of fundamental importance in therapeutics and in prognosis. The recognition of the ability of the heart to properly perform its functions depend largely upon the careful observation and study of the patient. Graphic methods, whereby disturbances of excitability, contractility, tonicity, conductivity, and rhythmicity are recognized, are splendid aids, but unless properly interpreted are worse than useless, because they may demonstrate alterations in one or more of these functions which may be taken as *prima facie* evidence that the heart is functionally insufficient when in reality it is not. Similarly the finding of a murmur in a routine diagnosis may lead to the erroneous conclusion that the heart is insufficient simply because the murmur is heard. Mackenzie says that only when a heart muscle is degenerated is there any loss of functional power. Murmurs and irregularities are usually only of significance when associated with some manifest impairment of the heart muscle and the influence of a murmur can only be interpreted by careful search for muscle impairment. However some irregularities, like auricular fibrillation, are of value in showing myocardial weakness; murmurs rarely, if ever, are evidence of degeneration. Increase in the size of the heart is a valuable sign. In functional dilatation changes in the size are unimportant unless accompanied by persistent increase in the rate of the heart and marked loss of power. The most valuable signs of insufficiency of the heart, however, are the subjective symptoms of the patient when called upon to do certain purposeful acts, as walking up or down stairs rapidly. Their interpretation requires a special nicety of judgment, as so many subjective symptoms are connected with the circulation that careful discrimination is required to find these evidences of exhausted muscle. Furthermore, it must not be forgotten that acts, which a laborer with a functionally weak heart can perform,

frequently cause grave signs of cardiac insufficiency when performed by a man with a functionally capable heart unused to labor or exercise.

One of the most valuable specific tests for cardiac power is that of Graupner. The blood pressure is estimated and the patient then walks up stairs rapidly. The pressure is again taken and a third observation made in about twenty minutes. Normally there should be a preliminary rise of 5 to 15 mm. Hg., followed by a corresponding fall by the time of the third estimation. In insufficient hearts there is a primary fall which continues at least until the third estimation is made. Other specific tests include the postural change in pulse-rate, shown by the fact that when a normal individual lies down the pulse-rate decreases six to twenty beats to a minute while if the individual has an insufficient heart the rate is unchanged. The time it requires a heart to regain its normal rate after a certain amount of work has also been suggested as a test of functional capacity of the heart. Hirschfelder says that the only accurate criterion of cardiac efficiency is whether a given strain causes it to diminish in size or dilate.

PART II

SPECIAL DIAGNOSIS

CHAPTER XXXI

THE SPECIFIC INFECTIOUS DISEASES

THE specific infectious diseases are those diseases which are caused by the invasion and growth in the body of specific pathogenic microorganisms, some of which are known, and some of which are not as yet isolated. The diseases due to vegetable microorganisms, or presumably so, are alone discussed in this chapter. The infections due to zoöparasitic organisms are included in the succeeding chapter.

The symptoms of an infectious disease are those (1) at the site of the invasion or the point of localization of the specific microorganism and (2) those that arise as a result of the liberation of toxins, including the general symptom of fever (*q. v.*). The severity of the symptoms depend upon the susceptibility to infection and the virulence of the causative pathogenic organisms. The infectious diseases may be *sporadic*, occurring in occasional scattered cases, *endemic*, occurring always in a few cases in certain definite localities, or *epidemic*, affecting many persons simultaneously and spreading rapidly to other places.

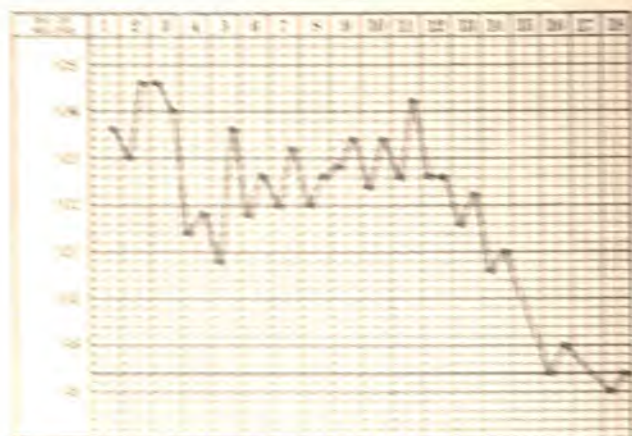
Typhus Fever.—An acute contagious disease of unknown origin, transmitted by the body louse, occurring sporadically, often becoming epidemic, characterized by abrupt onset with chill or with chilliness, a *rapid rise* of temperature, a peculiar spotted eruption, pronounced nervous symptoms, and by a crisis which occurs on or about the fourteenth day. Typhus fever is variously known as *ship fever*, *jail fever*, and *camp fever*.

Symptoms.—The period of *incubation* is usually about twelve days; malaise may precede by a day or two the onset of the disease. *Invasion* is characterized by headache, faintness, chilliness, or a distinct rigor, pains in the back and thighs, and extreme weakness. The *pulse* is frequently 100 to 140, the patient is restless and sleepless, and is annoyed by tinnitus. The *expression* of the flushed face is listless and dull.

About the fourth or fifth day the typhus eruption begins to appear. It consists at first of dull red macular spots of irregular size and shape. They are most numerous on the covered parts. In a few days these

become petechial. The skin is also mottled between the spots. Purpura and hemorrhages are sometimes met with in severe cases. The eruption does not occur in successive crops. When the disease is fully developed the face is flushed, the conjunctivæ red, the pupils contracted, so as to resemble pin-holes (ferret-eye), the tongue dry and brown, the teeth covered with sordes, the skin dry, hot, and stinging to the touch. Headache has given place to delirium, which may be wild and fierce, but is more commonly low and muttering. There are marked nervous symptoms—subultus tendinum, tremors, and picking at the bedclothes. The stupor may gradually clear up, or, on the other hand, deepen into coma; or the patient may die from progressive weakening of the heart, with or without pulmonary complications. In the majority of favorable cases, on or about the fourteenth day, a typical crisis occurs.

FIG. 162



Typhoid fever typical. (Day.)

Complications.—The most important complications are hyperpyrexia, laryngitis, bronchitis, congestion of the lungs, bronchopneumonia, septicæmia, heart failure, parotitis or other inflammatory glandular swellings, septic arthritis, gangrene of the smaller extremities, and edema of the pottis occur at times.

Laboratory Diagnosis.—The blood usually shows a leukocytosis only in the latter stages of the disease. The urine is scant, high-colored, with some albumin and mucus. Stranguria occurs not infrequently.

Diagnosis.—In sporadic cases only is the diagnosis fraught with difficulty. The rapid onset, the heat, and the acute nervous symptoms are most suggestive.

In the differential diagnosis, *cerebrospinal fever* is distinguished from typhoid fever by greater and more persistent retraction of the head and by a

result of lumbar puncture, and by the absence of the macular and petechial eruption.

Typhoid fever is distinguished by its slow onset and marked abdominal symptoms. The eruption of typhus is petechial and comes out on the fourth or fifth day; that of typhoid fever consists of rose spots and appears on the seventh or eighth day in distinct crops. In typhus fever the severe initial chill, the sudden onset, the greater prostration, and the earlier appearance of cerebral symptoms are helpful in distinguishing it from typhoid fever. The serum reaction must be employed. Typhus fever at the onset may also possibly simulate smallpox, measles, and scarlet fever, but in a few days differentiation is usually readily accomplished.

Clinical Types.—*Typhus siderans* is a malignant form of the disease, death taking place in the course of a few days.

Tabardillo (Mexican typhus) is a form of typhus found in Mexico in which the onset and defervescence of the disease is much slower than in the European type.

Mild Typhus.—Brill has described a clinical syndrome which was for a time believed to be a specific disease, but the weight of evidence at present seems to be to regard this as a mild or attenuated form of typhus fever.

Rocky Mountain Spotted Fever.—An acute infectious endemic disease, transmitted by a variety of wood-tick found in the far Western States of Idaho, Montana, Wyoming, and Nevada, and characterized by gradual onset, a macular, becoming petechial, eruption, and irregular fever.

Symptoms.—The incubation is from five to ten days and is succeeded by a severe chill, severe muscular pain, epistaxis, and malaise. The temperature rises rapidly and its range is usually high and distinctly irregular. About the third week it reaches normal or becomes subnormal. On the second to fifth day the rash appears on the wrists and ankles, gradually spreading over the entire body becoming especially thick on the back. At first rosy-red the spots soon become petechial, gradually fading as the temperature returns to normal. The intervening skin is jaundiced and cyanotic. In severe cases gangrene of the smaller extremities, scrotum, or penis may be grave complications.

Diagnosis.—The disease is usually readily differentiated from typhoid and typhus fever, or in children from cerebrospinal fever.

Variola.—Variola, or smallpox, is an acute contagious fever, beginning abruptly with chill, and followed in two or three days by a characteristic eruption which starts as discrete papules, soon becoming vesicular and later pustular.

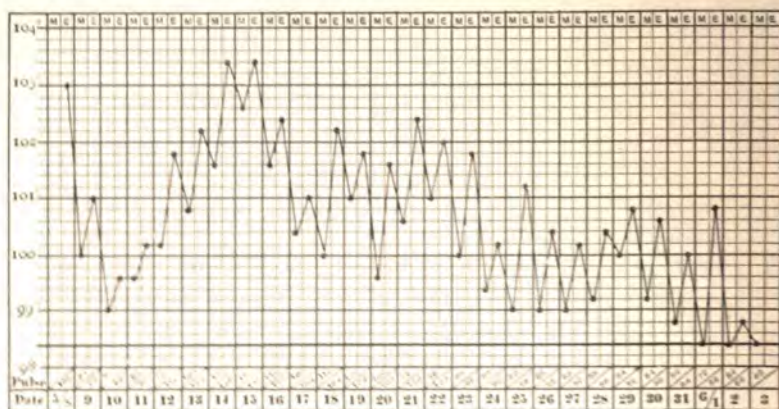
Symptoms.—For convenience of description the disease may be divided into three stages: (1) invasion, (2) eruption, (3) desquamation.

After an incubation period of ten to fourteen days, the *invasion* comes on abruptly and is marked by chilliness or a distinct rigor, excruciating headache, severe pain in the lumbar regions, and some-

times delirium or convulsions, especially in children. The temperature usually rises rapidly to 104° F. or higher in the first twenty-four to forty-eight hours, then to fall almost to normal, to remain there until the appearance of the pustular stage (Fig. 163). Prostration is extreme. Erythematous eruptions are not uncommon, especially on the inner surface of the legs and thighs. Petechiæ are found in Simons' triangle, the base of which is at the umbilicus and apex at the knees.

The stage of invasion lasts generally three days; but it may be shortened to two in very severe cases or lengthened to four in very mild ones, and in complicated and hemorrhagic cases it merges into the stage of eruption.

FIG. 163



Temperature in smallpox Adult: mild case. (Original.)

Eruption.—The characteristic eruption of smallpox appears first as minute specks resembling flea-bites. These in two or three days develop into small papules which feel like shot under the skin. In a day or two more the papules become vesicles, at first containing a clear fluid, which, however, rapidly becomes turbid; they are umbilicated. In the course of another day or two the vesicles have become pustules and are globular in shape. The eruption appears on the forehead, along the margin of the hair, and in the scalp, subsequently progressing over the rest of the body from above downward. The eruption is most abundant upon the face and hands, often being confluent here when discrete elsewhere. This period, when pustulation is at its height, lasts about three days; it is characterized by a marked secondary fever, the temperature rising as high as, or higher than, in the onset of the disease.

Desquamation.—The temperature now starts to subside, reaching normal about the eighteenth day. The pustules begin to dry up (desicca-

tion) and form dry scales or scabs, which are cast off toward the end of the third week of the disease; when the pustules have been sufficiently deep to involve the true skin, characteristic scars, called pits, are left.

Varieties.—Four varieties of variola, are recognized: (1) discrete; (2) confluent; (3) hemorrhagic; (4) varioloid.

In *discrete* variola the pocks are not numerous, and are separated from each other by intervening healthy skin.

In *confluent* smallpox the pustules are close-set, occupy almost the whole body, and coalesce, so that the face looks as though covered with a black, rough mask; the mucous membranes are also covered. The symptoms of the invasion are intensified, and the eruption may appear before the third day. Patients are liable to suffer with profuse salivation, uncontrollable vomiting, or diarrhea (especially children), and with delirium, which is often violent and destructive. The face is dreadfully swollen and the eyelids may slough; the feet and limbs also may be swollen and painful. There may also be severe bronchitis and pneumonia, abscesses, extensive sloughing, and a pyemic condition.

Hemorrhagic.—Two forms of hemorrhagic or malignant smallpox are recognized: (1) the purpuric or true hemorrhagic form, and (2) the vesicular hemorrhagic form.

True hemorrhagic or *black smallpox* is characterized by the occurrence of hemorrhages under the skin and bleeding from the mucous membranes. The mind remains acutely clear and the patient is conscious of his terrible condition. Death almost always takes place on the fourth to sixth day, usually before the appearance of a true eruption.

In the second form, hemorrhages take place into the base of the papules and into skin intervening between them. Death usually occurs before the appearance of the pustular stage. This form must not be confused with those cases of smallpox in which hemorrhage accidentally takes place into a few individual pocks.

Both these forms are accompanied by great prostration and severity of symptoms. The rash is always confluent.

Varioloid is a mild form of smallpox occurring in a person partially protected by vaccination, or in a person who, from other causes, does not possess the average susceptibility. The initial symptoms, as a rule, are as severe as in ordinary smallpox. Prodromal eruptions, especially the erythematous, are very common. The discrete eruption passes from the vesicular stage, as in ordinary smallpox; but here the process, as a rule, ceases, the vesicle drying up on the fifth or sixth day of the eruption. If pustules form, they do not reach their full development.

Laboratory Diagnosis.—Councilman and others have demonstrated the presence of a protozoön, the *Cytorrhycles variolæ*, in the epithelial cells of the affected parts. They are found with difficulty in stained sections only. The blood shows a leukocytosis of the mononuclear cells.

Diagnosis.—The diagnosis depends upon the history of exposure or the presence of an epidemic, the severe initial prostration, the fever course, and the eruption.

Differential Diagnosis.—When fully developed, smallpox will not be mistaken for any other disorder. In the initial stage, however, there may be doubt whether the disease will prove to be pneumonia, cerebrospinal meningitis, or typhus.

In *pneumonia* excruciating backache is wanting, and, on the other hand, the respiration is increased out of proportion to the pulse, and even in this early stage there may be cough and roughening of the respiratory murmur on one side.

Typhus fever begins abruptly with chill and high temperature; but the eruption which comes out on the fourth or fifth day is first macular and later petechial; the temperature does not fall with the appearance of the eruption.

Cerebrospinal meningitis and smallpox can be accurately differentiated by a lumbar puncture.

Measles.—The atypical cases of smallpox and measles or chickenpox may at times be mistaken for each other.

In the papular stage of the eruption smallpox may be mistaken for measles; but the eruption of measles is relatively flat, smooth, and velvety; that of smallpox is acuminate, hard, and shot-like. The temperature in smallpox falls as the eruption appears; that of measles remains high and even increases.

Chickenpox.—In children the vesicular stage of varioloid may be mistaken for chickenpox. In the latter the eruption is practically vesicular from the start, occurs without prodromata, appears in crops, is usually very scanty, and rarely becomes umbilicated or pustular. There are, however, severe forms of varicella, in which fever, restlessness, and cough precede the appearance of the rash, which is copious, some of the vesicles being inflamed at the base, some umbilicated, and some with purulent contents. In such cases the diagnosis cannot always be made from the eruption, but the history of exposure to one or the other infection must be considered, and the general clinical course must be observed.

Vaccinia.—The constitutional reaction that follows the inoculation into man (vaccination) of the virus of cowpox is known as vaccinia. Vaccination produces a local poek which first appears as a papule on the third day. By the fifth or sixth day this has gradually increased and become a round vesicle, the margins of which are raised and hard, the centre sunken (umbilicated). By the tenth day the vesicular contents have become pustular and surrounded by a wide areola which starts to fade about the twelfth day. The lymph begins to dry about this time so that at the end of the second week there remains a brownish scab which becomes dry and hard, falling off at the end of the third week and leaving a round pitted scar.

The constitutional symptoms consist of slight fever, appearing about

the third or fourth day, which may persist for four or five days; *malaise* and irritability, especially in children, and a marked *leukocytosis*. The neighboring lymph glands may be enlarged and tender.

The immunity against smallpox, conferred by vaccination with cowpox, is complete in about three weeks and persists for a variable time, usually about eight or ten years.

Varicella.—An acute, infectious fever, occurring almost exclusively in children, and characterized by the appearance, in successive crops, of colorless or pearly vesicles which dry up and are shed in from two to five days. It is attended with very little constitutional disturbance. A second attack is extremely rare.

Symptoms.—The *incubation* is generally about two weeks, but may be one or three weeks. In ordinary cases the first evidence of the invasion of the disease is the appearance of the eruption. In other cases, the severer ones, the child may be noticed for some hours or several days to be indisposed, and complain of loss of appetite, nausea, headache, and vague, muscular pains. The *fever* is almost always moderate—100° to 101° F.

FIG. 164



Varicella on the fifth day of eruption. (Welch.)

Eruption.—The eruption consists first of hyperemic macules. These macules rapidly become first papules and then vesicles. The papules are not hard as in variola. They appear at first upon the chest, neck, face, and scalp, then upon the trunk and limbs, and are usually widely scattered, being more numerous on the back. The development of the vesicles is so rapid that the eruption appears vesicular from the start. The vesicles vary in size from that of a pin-head to that of a small pea. They are very superficial, and usually rest upon a base that is slightly or not at all hyperemic. The contents are at first watery, and subsequently become pearly. Distinct umbilication is rare, and pustulation still more rare, but both occur. Sometimes the vesicles are to be seen upon the buccal mucous membrane and upon the throat.

SPECIAL DIAGNOSIS

While most of the eruption appears on the first or second day, fresh vesicles continue to appear for several days. In scrofulous and badly nourished children the lesions are more inflammatory and pustules are more common. If they are scratched, ulceration ensues. Hemorrhagic and gangrenous forms have been described. In ordinary cases, during the eruption the child is rarely more than indisposed; complications are rare, and the prognosis most excellent.

Desiccation.—Desiccation usually occurs by the fourth or fifth day, and may be present in the first day or two. The vesicles dry up and form yellowish or brownish scabs which drop off, leaving a slightly reddened spot. As the eruption appears in successive crops, all stages, from the initial macule to the dried scales, can often be seen in one case.

Diagnosis.—This is based upon the history of exposure and the eruption, appearing in crops. The laboratory offers no aid.

Varicella is distinguished from *vesicular* and *pustular eczema* by the fever, the symmetrical grouping and discrete character of the lesions, the comparative absence of itching and burning, and its shorter course.

Impetigo is distinguished by the absence of fever, the more local character of the eruption, and the fact that it is generally pustular. The eruption is more common upon the face and hands than in varicella.

Scarlet Fever.—Scarlet fever is an acute, contagious disease of unknown etiology, characterized by a sudden onset, with vomiting, sore throat, and high fever, followed in twelve to twenty-four hours by a bright red, punctiform eruption, by a very frequent pulse, by a desquamation which is often in large flakes, by a very variable degree of severity, and by a large number of complications and sequelæ, especially nephritis and inflammation of serous membranes. It preferably affects children from one to five years of age.

Few diseases vary so greatly in severity in different cases and in different epidemics. It may either be the mildest or the most malignant of diseases.

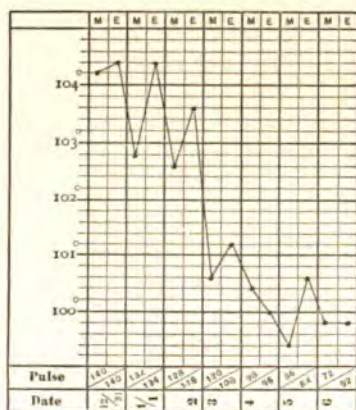
Symptoms.—The period of *incubation* is remarkably short, generally from three to five days; but it may be only a few hours, and in exceptional cases six days.

Invasion.—The invasion is abrupt. It is very common to be told that a child was apparently well on going to bed, but awoke in the middle of the night vomiting profusely and complaining of sore throat. The child is found in the morning with a temperature of 103° to 104° F., a pulse of 120 to 140, and a scarlatinal eruption beginning to show upon the neck and upper part of the chest. Close observation in such cases might have discovered that the child was feverish on going to bed and somewhat chilly before that. Onset with decided chill, vomiting, and nervous symptoms indicates a severe case.

The *subjective symptoms* of scarlatina are few; they consist of pain in swallowing, with stiffness of the neck muscles, some headache, thirst, malaise, and a moderate amount of weakness. In the early stage the skin itches, burns, and is frequently hyperæsthetic.

The *objective symptoms* and their order of succession are very characteristic. Vomiting is the rule, except in mild cases, and hence is of importance in diagnosis, especially in otherwise doubtful cases. The temperature is high at the onset, frequently 103° or 104° F. It falls a degree or so in the morning; but the following evening, when the eruption is usually at its height, it rises to 104° or 105° F., and then gradually falls to normal in the course of a week in ordinary cases. The *pulse-rate* is characteristically frequent, being 120 to 160 oftener than slower. This frequency is not an indication of danger. The *throat* exhibits a uniform flush extending over pharynx, tonsils, soft palate, and sometimes forward on the hard plate, nearly to the teeth. Sometimes dark red points can be distinguished on the soft palate. The tonsils are inflamed and projected toward the median line from each side. It is not uncommon to find a severe follicular tonsillitis at the first visit.

FIG. 165



Scarlet fever. Mild attack; intense eruption. (Original.)

The *tongue* is at first covered with a thick, creamy fur, through which enlarged red papillæ show. The enlarged papillæ look like small grains of red pepper sprinkled on the tongue. Sometimes the papillæ are elevated and have a button-like appearance. The symptoms appear very early in the disease, and may continue for three or four weeks. The coating soon disappears from the tip, leaving it bright red—the "strawberry tongue."

The characteristic *eruption* usually appears within twenty-four hours of the onset. Sometimes it comes out very slowly, seeming to be just ready to appear, but not appearing in its full development for four or five days.

The intensity of the *eruption* varies from a scarcely perceptible erythema to the color of a boiled lobster, and is proportionate to the severity of the disease. In ordinary cases the patient appears to be

covered with a uniform red efflorescence; but a closer inspection shows that there are darker red spots, between which the skin is more erythematous. It is first seen about the ears and neck, and spreads with great rapidity, covering the entire body in a day. It is more intense upon the trunk and flexor surfaces. Upon the extensor surfaces the punctate character is better seen. Pressure causes the redness to disappear, but it immediately reappears. Papular and vesicular lesions of eruption are also seen. The physiognomy is scarlet fever in peculiar. The circle about the eyes, nose, and lips remains pale, and in marked contrast with the rest of the fiery red face. Itching and burning are annoying symptoms at times. The eruption fades gradually, in ordinary cases, when there is no pressure or irritation, disappearing toward the end of the week.

The eruption is succeeded by *desquamation*, which is extensive in proportion to the intensity of the eruption. The flakes are larger in measles, and in severe cases the epidermis may come off in strips. This shedding from the hands and feet is sometimes so great that the cutaneous cast is comparable to a glove. This stage may be protracted for several weeks, danger of infection lasting as long as desquamation continues.

Varieties.—In addition to the ordinary form already described, scarlatina exhibits many irregular forms. There may be only a throat or follicular tonsillitis. If a rash is present, it is very faint, hence easily overlooked (*abortive type*). The diagnosis in such cases must be made from the fact of exposure to infection and from the appearance of the throat. The occurrence of vomiting is not important in the diagnosis, as it is rare in ordinary pharyngitis or tonsillitis.

Severe diarrhea may prevent the eruption from developing upon the skin. It appears upon the fauces, and the diagnosis is based upon this, the pulse and temperature, and the fact of exposure.

In *scarlatina anginosa* pain in the throat is great and deglutition is difficult. The tonsils are greatly swollen so as almost to occlude the fauces, and their surfaces are covered with creamy exudate. The cervical glands are swollen, and there is a tense and brawny cellulitis. Sometimes the tonsils become gangrenous, and the cervical or maxillary glands suppurate or become gangrenous, with resulting pyrexia and death. Suppuration may extend to the ears and axillary sinuses. In this form, also, a false membrane (*streptococcic*) is sometimes formed on the fauces.

In *malignant* forms the attack is ushered in with a chill, followed by hyperpyrexia, convulsions, marked ataxic symptoms, or stupor. Some patients lie in coma-vigil, others are very restless and delirious. Death in a few days is the rule. In rare cases death takes place in a few hours, without the appearance of any eruption, the *fulminant type*. In other cases hemorrhages into the skin are seen and there is hematuria and epistaxis (*hemorrhagic form*). Death occurs in sev-

days. In still other rare cases the symptoms of septicæmia develop and the patient dies in the second or third week from pronounced toxæmia (*septicæmic type*).

Complications and Sequelæ.—Of these, nephritis and otitis media are the most common, one or the other occurring in about 22 per cent. of all cases. Less frequently there may occur an endocarditis or pericarditis, a pneumonia or a pleurisy, a peripheral neuritis or a meningitis, an arthritis, or very rarely a suppurative adenitis or a cancrum oris.

Laboratory Diagnosis.—There is a hyperleukocytosis (20,000 to 30,000) of the polynuclear type, reaching its highest point about the third day and then gradually declining to normal in about fourteen to sixteen days. The urine is the ordinary febrile urine unless nephritis develops (*q. v.*).

Diagnosis.—Sudden onset, rapid rise of temperature, persistent and causeless vomiting, and sore throat lead one to suspect this affection. The characteristics as well as eruption and its mode of evolution, a punctate eruption in the axilla and in the groins, the rapid pulse, the peculiar tongue, the circle of pallor on the face, are all characteristic of the eruptive stage.

Differential Diagnosis.—Measles, rubella, acute local faucial infections, acute gastritis, acute meningitis, pneumonia, and diphtheria must be differentiated from scarlet fever at times.

Measles.—Scarlet fever is distinguished from measles by the mode of onset, which is sudden, with chilliness, high temperature, vomiting, sore throat, great rapidity of the pulse, and leukocytosis, whereas the onset in measles is gradual, with coryza, cough, moderate fever, perhaps looseness of the bowel but no sore throat. The eruption of scarlatina occurs on the first day, that of measles on the fourth; the former consists of dark red spots, with intervening erythematous skin, the whole looking at a distance like a uniform bright red flush; the latter consists of raised, rounded, or flattened spots or blotches, velvety to the touch, and, upon the body and extremities, grouped in patches with crescentic outlines.

In ordinary *pharyngitis* and *tonsillitis* the redness is more apt to be confined to the pharynx, tonsils, and arches of the soft palate; in scarlatina it extends as a flush over the soft and hard palate and buccal surfaces. In the former, high temperature, a very frequent pulse, and vomiting are unusual; in the latter they are the rule.

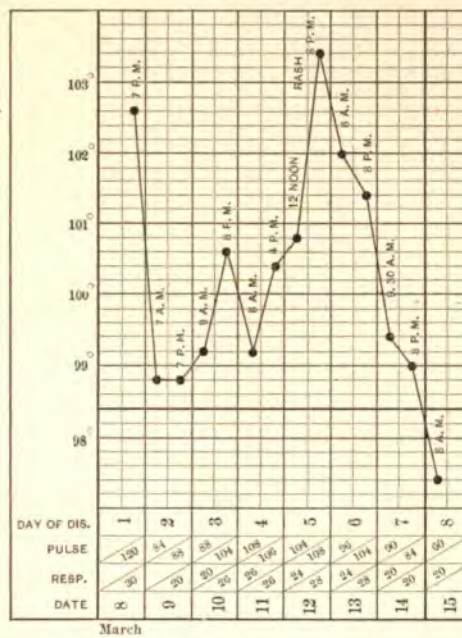
In *acute gastritis* there is usually a history pointing to indiscretion in eating, with constipation; sore throat is absent, and any erythema present lacks the characteristic rash.

The diagnosis from *rubella* is difficult at times. It differs from scarlatina in presenting mild catarrhal symptoms, sneezing, suffusion of the eyes, and cough, with a relatively fleeting eruption. The latter perhaps appears most frequently upon the back and chest. It more commonly resembles the rash of measles than that of scarlatina, but when it resem-



second day to normal or nearly normal, and then steadily rises until it reaches its acme with the full development of the eruption on the fourth to sixth day, when, in uncomplicated cases, it falls rapidly to normal. With the coming out of the eruption the coryza increases in severity, and cough is a prominent and annoying symptom, depending upon a catarrhal inflammation of the entire respiratory tract, from the nose to the bronchioles. Diarrhea is a frequent associated system.

FIG. 166



Measles. Characteristic chart. Female, aged twenty-seven years. (Original.)

The Eruption.—The eruption on the skin appears first about the neck, face, and wrists, and spreads in two or three days over the entire body. It is usually most copious upon the face, which is swollen, dark red in color, and closely set with papules, which are elevated, rounded at the summits, and feel velvet-like to the touch. When to this picture is added that of a severe coryza with mucoserous exudate, which often glues the eyelids together and oozes out upon the face, and a corresponding condition of the nasal orifices, the physiognomy is at once seen to be unusual. At this stage, moreover, photophobia is often considerable, the child burrowing its head in the pillow to escape the light.

The eruption is not apt to be confluent upon the body; here the dark red, elevated, smooth papules are very distinct. Sometimes they are grouped so as to form crescentic outlines. The eruption fades in order in which it appeared, and is followed by a fine branny

desquamation. With the completion of the eruption the fever falls rapidly to, or below, normal, the coryza and bronchitis improve correspondingly, and in forty-eight hours convalescence is fully established.

Complications.—Laryngitis, bronchitis and bronchopneumonia, ocular complications and otitis media are the more usual complications; more rare are noma, stomatitis, adenitis, and nephritis.

Convulsions may occur as a complication especially when pneumonia is developing.

Sequelæ.—In cases in which there has been severe diarrhea, measles is sometimes followed by considerable weakening of the digestive power, and even by a peculiar form of enteritis which may terminate fatally. The catarrh of the respiratory tract, which almost invariably accompanies it, predisposes to the development of whooping cough and tuberculosis. Paralysis may follow measles.

Varieties.—Measles without catarrh is rare. It cannot be distinguished from a measles-like rash, seen in r  theln, except by the occurrence in the neighborhood of other cases of undoubted measles. Measles without eruption is to be recognized by the coryza, possibly with eruption on the soft palate, the course of the temperature, and the exposure to specific infection.

Black measles is the name given to malignant forms in which the skin is dusky and the eruption comes out poorly and has a bluish color. In rare instances the eruption shows a hemorrhagic tendency, the spots being livid or ecchymotic. Actual hemorrhages from mucous surfaces may occur, the patient dying in coma or convulsions.

Laboratory Diagnosis.—The presence of a leukopenia is a valuable diagnostic aid in recognizing and differentiating measles from other exanthemata. The urine usually shows the diazo reaction.

Diagnosis.—The diagnosis is based upon the history of exposure, Koplik's spots, the coryza, the characteristic fever range, and the eruption.

Differential Diagnosis.—Measles must be differentiated at times from scarlet fever (*q. v.*) or from rubella. In the latter condition the coryza is not so marked; Koplik's spots are absent; the symptoms are mild and the rash appears early.

Rubella.—Rubella (r  theln—German measles) is an acute, contagious fever, characterized by a gradual onset, with moderate fever, sore throat, slight coryza, and eruption.

Symptoms.—The incubation period varies from one to three weeks, but is generally about two.

The invasion is without prodromata, or no more definite than languor and indisposition, the first thing noticed being the eruption. This in some cases consists of pale red, smooth, slightly raised blotches, closely resembling measles, but more pronounced on the trunk, and discrete. More commonly it consists of rose-red macules or papules, occasionally confluent, but usually discrete, and most marked upon the trunk. In still other cases the eruption closely resembles that of

scarlatina, differing chiefly in being a paler red and accompanied by less heat of the skin.

Sometimes the eruption is circumscribed, as upon the face or limbs. It is usually the seat of considerable itching, and this may be the first symptom that attracts the patient's attention. It will be seen that the eruption is multiform in character. Concurrently with the eruption there is usually slight rise in temperature (100° to 101° F.), suffusion of the eyes, with slight lacrimation and photophobia, and slight pharyngitis; nausea is not uncommon, but vomiting is very rare.

The eruption extends over the body in twenty-four to thirty-six hours, and pales quickly, fading on the portions of the body first attacked before reaching its height on the last, and being completed in three or four days. Sometimes a branny desquamation succeeds.

In addition to the mild coryza and eruption, the most important objective symptom is swelling of the cervical glands, all of them being sometimes swollen, especially those behind the sternomastoid, the auricle, and along the margin of the hair.

Rubella has few complications: bronchitis, pneumonia, and otitis occur rarely, and still more rarely false membrane on the throat, and albuminuria. It ends almost invariably in recovery, except in very feeble children.

Laboratory Diagnosis.—Normal total leukocyte count with relative lymphocytosis.

Mumps.—An acute infectious disease, characterized by swelling of the salivary glands, especially the parotid.

Symptoms.—Invasion is sudden, with chilliness, a rise in temperature which is generally moderate (101° to 103° F.), and pain at the angle of the jaw. The corresponding parotid gland as well as the adjacent cellular tissue begin to swell rapidly. The swelling is tender and boggy and can be observed in front of the ear extending below and around it and behind the ramus of the jaw. Along with pain on movement of the jaws, any acid liquid, as vinegar, which stimulates salivary secretion, increases the pain. At times the submaxillary glands are involved instead of the parotids, or they may be enlarged and painful several days before the parotid is affected. The disease may be limited to one side or involve the opposite side, as the process in the one first attacked subsides. Rarely it is bilateral from the start. When the swelling has lasted from three to five days, the fever subsides and the swelling begins to disappear rapidly. At this time, however, the opposite side may be attacked or the testicles become inflamed. Usually it is the right testicle. Rarely in girls and women the ovary or mammary gland is inflamed. Resolution is extremely rapid, and usually the disease is not followed by sequelæ. Sometimes, however, deafness remains, or atrophy of the affected testicle.

Glandular Fever.—Glandular fever is a contagious disorder, the cause of which has not been accurately determined, occurring in children between the age of five and eight years. It is characterized by fever,

usually occurring abruptly, with headache, pains in the limbs and in the lymph glands of the neck. On examination of the fauces a slight pharyngitis is observed and the tonsils are enlarged. The rise of temperature is accompanied by frequent nausea and vomiting. The temperature rises abruptly to about 102° F. In the second twenty-four hours the glands of the neck, particularly those behind the sternocleidomastoid muscles, enlarge and are tender. Although there may be some slight edema, there is no redness or swelling of the skin. The fever continues for three or four days, the glandular enlargement, however, may persist for several weeks, and may end in suppuration.

The other lymphatic glands about the neck and in the axilla and groin may be enlarged. Commonly there is enlargement of the spleen and liver. The absence of an eruption serves to determine the infection from the eruptive fevers associated with adenitis, particularly measles and rubella.

Whooping Cough.—A specific catarrhal inflammation of the respiratory passages, involving especially the trachea and bronchi, caused by the *Bacillus pertussis* of Bordet-Gengou, and characterized by paroxysms of cough, which are succeeded by spasmodic closure of the glottis and a peculiar inspiratory whoop. The disease occurs especially in children under six years of age, is contagious and is sometimes epidemic.

Symptoms.—Whooping cough may be conveniently divided into three periods:

1. The catarrhal stage.
2. The spasmodic stage.
3. The stage of gradual subsidence of the disease.

First Stage.—The patient appears to have an ordinary cold, with a certain amount of redness of the mucous membrane of the eyes, nose, and throat. The cough is dry, and sometimes a ringing quality can be detected. The patient is irritable, has slight fever, diminished or capricious appetite, and restless sleep. A mild bronchitis of the larger tubes can be detected by physical exploration.

Second Stage.—Transition from the first to the second stage occurs in about ten days and is marked by the appearance of the characteristic whoop. The paroxysmal cough is made up of a series of rapid expiratory efforts, diminishing in force and duration; when these cease, there succeeds a prolonged crowing inspiration—the whoop. There may be only one paroxysm of coughing at a time, but more commonly, and always in severe cases, one paroxysm is succeeded by another. During the coughing the child's eyes become suffused, and there is a discharge of serum or mucus from the nose, and of saliva and bronchial secretion from the mouth. The face becomes swollen and dusky. If the child is walking about, it grasps some object for support during the paroxysm, which usually terminates in vomiting. During severe paroxysms, hemorrhages are apt to occur; these are generally small and most frequently submucous. In well-marked cases, when the disease has

lasted some time, the face has a characteristic appearance: it is swollen, sodden, and dusky, with dull, heavy, red, and watery eyes.

The number of paroxysms varies from two or three to twenty or thirty or more in twenty-four hours, and they are worse at night. The whoop, while characteristic, is not present in every case, being absent especially in babies and very young children. Sometimes children have choking spells without much coughing and without the whoop. Again, when pneumonia or measles occurs as a complication, the whoop usually ceases for the time, but may reappear later.

Third Stage.—The third stage is less well-defined than the first two. The number of paroxysms during the day diminishes, vomiting is a less frequent accompaniment, the appetite improves, the child gains in flesh and passes more restful nights. The duration of the disease is variable. Ordinarily it lasts from six to eight weeks, but it may be prolonged for several months.

Complications and Sequelæ.—Bronchitis, bronchopneumonia, atelectasis, acute transient emphysema and rupture of the lung occur relatively frequently as pulmonary complications; tuberculosis, chronic enlargement of the bronchial glands, chronic bronchitis, and persistent emphysema as sequelæ.

Hemorrhages (epistaxis and subconjunctival), nephritis, convulsions, ulcer of the frenum, digestive disturbances, prolapse of the rectum, hernia, and dilatation of the right heart are other more or less serious complications.

Laboratory Diagnosis.—The *Bacillus pertussis* of Bordet and Gengou can be demonstrated in the sputum. The blood shows a moderate lymphocytosis, persisting for weeks after the subsidence of symptoms.

Diagnosis.—With the appearance of the characteristic whoop the diagnosis is assured.

Rheumatic Fever (*Acute Articular Rheumatism*).—An acute infectious disease, characterized by specific inflammation of the joints and contiguous structures, and further by a tendency of the inflammation to involve the larger joints successively, to skip from one joint to another, and to be associated with endocarditis or pericarditis or more rarely with inflammation of other serous membranes. The tonsils act as a portal of entry for the exciting organism in most cases.

Symptoms.—The onset of the disease is not characterized by constant symptoms. Sometimes the fever and joint-inflammations are preceded a day or two by debility, wandering pains in the joints or muscles, tonsillitis, and loss of appetite. Other cases are marked by a chill or repeated attacks of chilliness, followed in a day or two by fever and inflammation of the joints. In rare cases the onset may be followed not by inflammation of the joints, but by inflammation of the serous membranes, particularly those of the heart and its sac.

The temperature may rise a day or two before there are any joint-symptoms, or fever and arthritis may begin almost simultaneously. The temperature rises rapidly to 102°, to 103°, or 104° F., and one or

more of the larger joints, generally the knee and ankle, become painful, tender, swollen, and hot.

THE JOINT.—There may be great pain on motion before there is evident swelling or much local tenderness. The pain varies from mere discomfort to the most excruciating suffering. It is always aggravated by motion or pressure, and is at times so exquisite that the lightest touch, or the weight of the bedclothing, makes the patient cry out. It may extend beyond the joint to neighboring tendons and nerves. The swelling likewise varies greatly; sometimes there is only slight puffiness with increased distinctness of the cutaneous veins, increased heat in the part, but no general redness; in other cases there is considerable swelling about the joint, so that the bony prominences are obliterated, the surface being tense, red, and very hot to the touch. There is not often effusion into the joint. Swelling is most marked in the knee, wrist, ankle, and less so in the shoulders, hips, and elbows. It is very common on the dorsal surface of the hand.

Multiplicity of Joints Affected.—A characteristic peculiarity of rheumatism is its tendency to involve one joint after another. The inflammation usually lasts from two to four days in each joint.* The process may subside in one articulation and begin in another with startling rapidity.

The *skin* does not feel so hot as one would expect from the temperature. It is continuously covered with a copious, acid, and somewhat pungent perspiration. *Nervous symptoms* are not marked. There may, however, be slight nocturnal delirium. Sleeplessness from pain is common.

The *temperature* in rheumatic fever is not usually very high; it is much oftener under than over 103° F. In rare cases, however, especially when the fever is complicated with pericarditis, pneumonia, or some disturbance of the heat-regulating apparatus, the temperature may attain the extraordinary range of 106° to 112° F. Such high temperatures may occur suddenly or gradually, and are sometimes attended with marked cerebral symptoms (so-called cerebral rheumatism).

The *pulse-rate* is moderately accelerated unless there is a cardiac complication, when it becomes very rapid.

Endocarditis and *pericarditis* may occur at any period of rheumatic fever; they may even precede the joint-inflammations. They are most common, however, in the first two weeks of the disease. The younger the patient and the more severe the attack, the greater the liability to heart-complications. They occur in about one-fourth of all cases. Endocarditis is most common; often it is the only lesion but sometimes it is associated with pericarditis and more rarely myocarditis. These complications usually give rise to no symptoms at first. Hence, the heart should be examined daily, and the pulse carefully followed.

Complications and Sequelæ.—Apart from heart-complications we have been mentioned, *pleuritis*, *pneumonia*, and *bronchitis* or

from 10 to 15 per cent. of the cases. They are frequently bilateral, and are very much more common in rheumatic fever with pericarditis or endocarditis than in simple rheumatic fever.

Nervous System.—The most common complication of the nervous system is *delirium*, which is generally associated with insomnia and hyperpyrexia, but the latter is not constant. These cerebral symptoms generally appear in the second week of illness, and about the time of convalescence, or while the joints are still inflamed. In favorable cases a deep sleep ushers in recovery; in unfavorable cases the delirium persists, the patient dying in collapse or coma, possibly preceded by convulsions.

Chorea sometimes occurs as a complication, but it is more common as a sequel of mild cases in children. *Cerebral meningitis* occurs occasionally, especially when there is ulcerative endocarditis. *Cerebral embolism* is another rare complication.

Various *spinal symptoms* occur in some cases, at times with and at times without demonstrable lesion of the cord or its membranes. Nephritis is rare, but sometimes hemorrhage into the kidney occurs with its usual symptoms.

Various *erythematous eruptions* are seen from time to time, and occasionally *purpura*. Subcutaneous nodosities have been described by several writers. They are attached to the tendons, fasciæ, and periosteum, and are most frequent on the back of the elbow, the ankles, and patella.

Laboratory Diagnosis.—The *urine* is of high specific gravity, strongly acid, and contains an excess of urates and uric acid crystals. The *blood* shows a hyperleukocytosis of the polynuclear type. A marked secondary anemia develops rapidly. Organisms of a streptococcic character have been frequently isolated by observers by blood culture and have been found in the focal lesions.

Diagnosis.—Rheumatic fever is diagnosticated by noting the profuse acid and acrid sweating, the tendency to involve a number of joints, and particularly the larger ones, the great intensity of constitutional symptoms and the great liability to heart-complications. It is differentiated from *gout* in which the joints involved are usually the smaller ones, by the above symptom-complex.

From *pyemia* it is distinguished by the wandering character of the inflammation; the acid sweats; the absence of any antecedent condition which would develop purulent foci, the absence of chills and sweating except after a fall in temperature. Cutaneous abscesses do not occur in rheumatism.

Acute synovitis resembles rheumatic fever, because in both occur symptoms of pain, tenderness, and swelling in connection with a joint. Usually, however, but one joint is involved in synovitis; the effusion is limited to the synovial sac, is frequently abundant and fluctuation can easily be detected. The *acute arthritis of infants*, which is pyemic in character and often of gonococcus origin, must be distinguished from rheumatism.

Acute periostitis when close to a joint simulates rheumatism; but the tenderness and heat are not in the joint itself; they are superficial and are associated with less swelling. Pitting on pressure is common; and circumscribed fluctuation usually discloses the presence of suppuration. Pyemic symptoms are added to the local symptoms, particularly if *osteitis* or *osteomyelitis* is present. The articular symptoms of *glanders* are to be distinguished by the presence of one or more pustules, and the fact that the painful joints are not so apt to be swollen and red as in rheumatic fever. In *syphilis* joint-pains frequently occur, but their character is made out by the fact that the joints are not inflamed, and that the pain is much worse, or occurs only at night. In *gonorrheal arthritis* the pain is constant in one or rarely more joints and the causative gonococci can be demonstrated by smears.

Subacute Articular Rheumatism.—In some instances the joint-inflammation is less severe, and is accompanied by only slight fever. One or more joints may be affected. Subacute rheumatism differs from the ordinary form in being milder in degree and more persistent, lasting sometimes for months. It is generally subacute from the beginning, but may represent a secondary modification of the type in those who have had several attacks of rheumatic fever and have been left in a very sensitive condition.

The type of rheumatic fever is usually subacute in children, and often only one joint is involved. Cardiac complications are more frequent than in adults, and chorea may occur as a sequel. Erythema nodosum and subcutaneous nodosities are more common in children.

Dengue.—An acute contagious disease, occurring in epidemics, characterized by severe pains in the head, back, and joints, by the presence of various skin eruptions, a prolonged convalescence, a very low rate of mortality, and caused possibly by a parasite transmitted by the mosquito (*Culex fatigans*).

The disease occurs in epidemics in tropical and subtropical countries, and rarely in cooler climates. In the southern parts of the United States an expressive name given to the diseases is break-bone fever.

Symptoms.—The period of incubation is short, varying from a few minutes to several days, or even a week. Invasion is sudden and is rarely preceded by prodromata. It is marked by chilliness or a chill, and very severe pains in the head, back, and limbs. In children the onset may be marked by convulsions, which are sometimes followed by stupor and vomiting. The pains are sometimes excruciating, and there is extreme debility. The temperature rises to 102° or 103° F., but rarely is much higher. The pulse is frequent—110, 120, or more. In from one to three or five days the temperature falls to or below normal (the remission), accompanied by sweating or diarrhea, and fluctuates about this level for several days, when a second and moderate rise in temperature, which is of short duration, occurs. During the first rise in temperature there is usually a transient erythema which is not followed by desquamation. During the remission eruptions—scarlatiniform,

herpetic, urticarial, or miliaria-like—begin to appear, accompanied by the secondary rise in temperature. The eruptions may come out in successive crops, and are followed by desquamation. Convalescence is now established, but may be interrupted by relapses. The most frequent complications are disorders of the nervous system, but bronchitis and diarrhea occasionally occur.

Laboratory Diagnosis.—There is a leukopenia associated with a lymphocytosis. The urine is usually normal.

Differential Diagnosis.—Yellow fever and dengue, in spite of the jaundice, the frequency of hemorrhage and the characteristically slow pulse of the former condition, are at times mistaken one for the other largely on account of the prevalence of both diseases at the same time.

General Gonorrheal Infection.—Although the infection is usually limited to the genito-urinary tract, the gonococcus may enter the blood and infect tissues elsewhere, causing a local inflammation. We therefore see symptoms due to the primary infection; symptoms due to the infection of the genito-urinary organs by direct continuity; and systematic infection. The primary infection involves the adnexæ of the genital organs in the male and the female. In both sexes, cystitis, ureteritis, and pyelitis secondarily occur. The infection is usually mixed. When the gonococcus invades the blood, symptoms of septicemia or pyemia arise. The infection may be rapid and fatal, and may terminate ten days after the primary lesion.

In other infections the joints become involved and we have the phenomena of gonorrheal arthritis (see Joints). Endocarditis, myocarditis and pericarditis may also occur.

Diagnosis.—A positive diagnosis is obtained when the organisms are found in the genital discharges or are isolated by blood culture. Irons employing intradermal injection of glycerin extracts of gonococci cultures, finds an anaphylactic skin reaction occurs in infected cases, in from twenty-four to forty-eight hours.

Differential Diagnosis.—Acute exacerbation of arthritis deformans, gout, acute articular rheumatism, and septic arthritis may simulate gonorrheal arthritis. Gonorrheal septicemia can only be differentiated from other septicemias by a positive blood culture, and the same is true of gonorrheal endocarditis; either, however, may be provisionally diagnosed if a focus of gonococcic infection is found.

Acute Infectious Jaundice (Weil's Disease).—An acute infectious disease occurring especially in butchers, laborers, and brewers, characterized by sudden onset and followed by pronounced jaundice, caused probably by the *Bacillus proteus* fluorescence, but also attributed to *Bacillus coli* infection, has been described by Weil.

Symptoms.—After exposure to cold generally, the patient is seized with a chill, followed by fever, with headache, vomiting, and epigastric pain. Jaundice sets in rapidly. The temperature remains high or may be intermittent. Stupor, at times violent delirium, and coma, albuminuria with suppression of urine, subcutaneous hemorrhages, and hemorrhages

from mucous membranes, rapidly ensue. In one of the author's cases there was enlargement of the liver, with subcutaneous edema over the hepatic area. The acute symptoms usually subside in from four to eight days, the fever in about two to three weeks, but the jaundice may persist for some time. Recovery is the rule.

Differential Diagnosis.—Typhoid fever, dengue, yellow fever, simple jaundice, and acute yellow atrophy must at times be differentiated from Weil's disease.

Miliary Fever (Sweating Sickenn).—An acute infectious disease, occurring in epidemics, and characterized by moderate fever, profuse sweating, tenderness, and a sense of oppression in the epigastrium, and a miliary vesicular eruption. The disease has occurred epidemically in England, but is not met with now outside of France and Italy. Miliary fever is distinguished from rheumatism by the moderate fever and absence of joint-swellings, and from malarial fever by the absence of chills, of periodicity in the febrile movement, and absence of malarial organisms from the blood. The duration of the disease is from one to four weeks. The mortality in some epidemics has been very high, in others very low.

Milk Sickness (Puking Fever).—An acute disease affecting cattle, and transmitted from them to the human being in milk or meat. The disease is limited to a few sparsely settled localities west of the Allegheny Mountains. It is characterized by great debility with muscular tremor upon motion, vomiting with a peculiar fetor of the breath, obstinate constipation, and moderate fever or subnormal temperature. The duration is usually less than a week. The patient may sink into a typhoid condition and die in coma, or he may die in a few hours. Convalescence is protracted.

Foot-and-mouth Disease (Lipidus Fever).—An infectious disease, communicated to man through cattle, sheep, or pigs, and characterized by a stomatitis. It is communicable by milk, or by direct contact. The etiological agent is unknown. The period of incubation is from three to five days. Invasion is characterized by slight fever, heat, and soreness of the mouth, and the development of vesicles which burst and leave shallow ulcers. The tongue swells greatly, eating is painful, and salivary profuse. The disease runs from one to two weeks, and ends almost invariably in recovery.

Encephalitis, Rabies.—An acute infectious disease caused by the virus of an unknown organism and communicated to human beings by the bites of animals similarly affected. The period of incubation is uncommonly long and very variable—from two weeks to two months usually. The disease has been divided into three stages—the prodromic, the spasmodic, and the paralytic.

In the prodromic stage there is pain, hyperesthesia, or numbness at the seat of the wound. The nervous system is extremely depressed in spirit and may be delirious. He is affected with thirst, but his attempts swallow water cause intensely painful spasms of the larynx.

The *second stage* is reached usually on the second day. The laryngeal spasms are increased and lead to intense dyspnea and to pitiable struggling and gasping on the part of the patient. In addition to the convulsive seizures, the patient foams and froths at the mouth, and his face expresses the extreme terror and mental anguish he feels. The second stage lasts from one to three days, and is followed by the *third stage*, exhaustion intermitting with paroxysms of less severity, together with paralysis of groups of muscles. The patient may now be able to swallow easily, but there is great weakness of the heart, and death may occur from failure of the heart, from asphyxia, or in a convulsion. The duration, as indicated, is only a few days. In the absence of specific antirabic treatment, the result is practically always fatal, but recovery may be possible.

Laboratory Diagnosis.—Examination of the central nervous system of animals suspected of having or causing rabies will show the widespread, irregularly shaped Negri bodies, varying in size from 4 to 10 μ . By this method the hysterical cases of pseudohydrophobia (lyssophobia) can be differentiated from true hydrophobia.

Anthrax.—An acute infectious disease, caused by accidental inoculation of the anthrax bacillus from living cattle or their products and characterized by cutaneous or visceral symptoms.

Cutaneous Anthrax.—External anthrax manifests itself in two forms, the malignant pustular—and much less frequently, the malignant edematous form. In the first form the period of incubation varies from a few hours to several days, the patient has a prickling or burning feeling on some exposed part of the body, particularly the hand, face or neck. At the seat of irritation, first a papule, then a vesicle develops. The vesicle may attain considerable size. The contained fluid quickly passes from clear to bloody, and then escapes, leaving a dark brown or black scab.

Instead of disappearing, the base of the vesicle becomes inflamed and indurated, the induration extending to surrounding tissue and causing a condition of brawny edema. A whole arm or one side of the face and neck may be swollen. There may or may not be an associated *lymphangitis* and *adenitis*.

The general health does not suffer at first, but in a day or two fever sets in, accompanied by delirium, sweating, great weakness, enlargement of the spleen, severe pains in the limbs, and diarrhea. Death, preceded by collapse, may occur in from five to eight days, except in the rare cases that terminate favorably.

In *anthrax edema* there is no pustule, but only a yellowish or greenish swelling of the tissues. Gangrene may ensue. It is seen most frequently in the eyelids, but may be on the head, hand, or arm.

Visceral Anthrax.—Internal anthrax attacks either the lungs or intestines. *Pulmonary anthrax* is characterized by intense dyspnea and a feeling of oppression or constriction. Breathing is labored, but not much accelerated. Only a few coarse rales are to be heard on

auscultation. The expectoration may be abundant and bloody, or absent. There is a tendency to collapse, with cold, bluish skin and a subnormal axillary temperature. The rectal temperature, however, is raised two or three degrees. Death may occur in coma and convulsions, or the patient may die suddenly, the mind being clear. The duration of the diseases is from one to five days.

Intestinal anthrax presents the following symptoms: the patient first complains of malaise, loss of appetite, pains in the limbs, giddiness, and headache. Then vomiting may set in, and a more or less severe diarrhea, the evacuations often containing blood. There may be pain in the abdomen, which becomes somewhat tumid; the spleen is enlarged. Epileptiform convulsions may occur; the upper limbs may be affected with tetanic spasms; there may be opisthotonos; and the pupils may be widely dilated. The pyrexia is slight, and death is preceded by extreme collapse. The duration of the disease is usually from two to seven days, but sometimes it is scarcely twenty-four hours.

Laboratory Diagnosis.—The *Bacillus anthracis* may be recovered from the blood by a blood culture or may be found in the pus from the pustule. The blood picture showed a neutrophilic leukocytosis in the few cases studied. Inoculation of the blood or pus into a guinea-pig, if the blood cultures are negative, will show the presence of the bacilli in large numbers in the guinea-pig's blood.

Diagnosis.—In suspicious cases occurring in shepherds, butchers, tanners, woolsorters, or in those whose occupation is connected with cattle, hides, or wool, the laboratory findings will readily differentiate the external form from a carbuncle, glanders, or a diffuse cellulitis. Internal anthrax is rarely diagnosed unless the infection is suspected and cultural methods are employed.

Typhoid Fever.—An acute, infectious disease, caused by the *Bacillus typhoeus*, characterized by a gradual onset, a continued fever, an eruption of rose-colored spots, marked nervous and abdominal symptoms, and an average duration of three or four weeks.

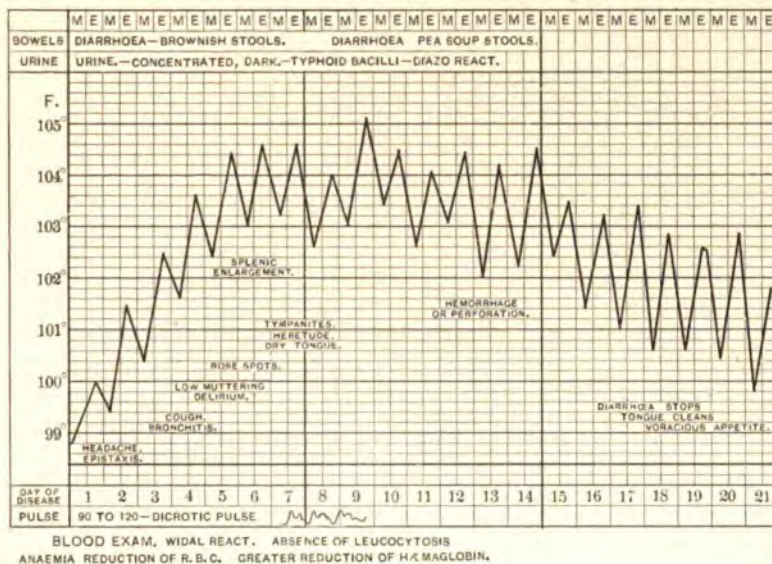
It occurs sporadically and epidemically, and in large cities is apt to be endemic. Its special habitat is in temperate climates, but it may occur anywhere. It is relatively rare in the southern and southwestern portions of the United States. It is more frequent in the latter part of the summer and in the autumn and winter and following hot and dry summer weather. Young adults are especially prone to it, but infection may occur at any age. The state of previous health does not seem to have any influence. All cases are the result of ingestion of polluted water (25 per cent.) or of transmission of the bacteria from the excretions of typhoid patients through the medium of flies, the hands, dirt, etc., to the food or milk.

Symptoms.—The period of incubation is commonly from one to two weeks. During this time the patient usually is languid, becomes tired easily upon exertion, has severe headache, and sleeps poorly. While the disease is in progress the onset is so muc

the rule that it becomes an important factor in the diagnosis from other disease conditions.

Invasion is not sharply marked. The beginning of fever is the most constant indications of the onset of the disease, and two very important early symptoms are cough from bronchitis, and epistaxis. The most prominent and constant subjective symptom during the first week is *headache*. Other very common symptoms are tenderness, rarely pain in the right iliac region, more or less prostration, and impaired appetite.

FIG. 167



Typhoid fever. Course of fever and relation to symptoms.

The *objective symptoms* are therefore the most important. The face is pale rather than flushed, and has a dull, listless, apathetic expression. The tongue is heavily coated with a white fur, which later becomes yellow. The abdomen is somewhat distended and tympanitic on percussion.

There is usually tenderness in the right iliac region, and gurgling upon palpation is pretty constant. Constipation may be present at first, and sometimes persists throughout the disease. A tendency to diarrhea is, however, characteristic of the disease. The number of stools varies from two or three to a dozen or more in twenty-four hours. They are light yellow in color (resembling pea-soup), thin, watery, and offensive.

Enlargement of the spleen is a very constant finding, usually first noticed from the fifth to the seventh day, and subsiding during convalescence.

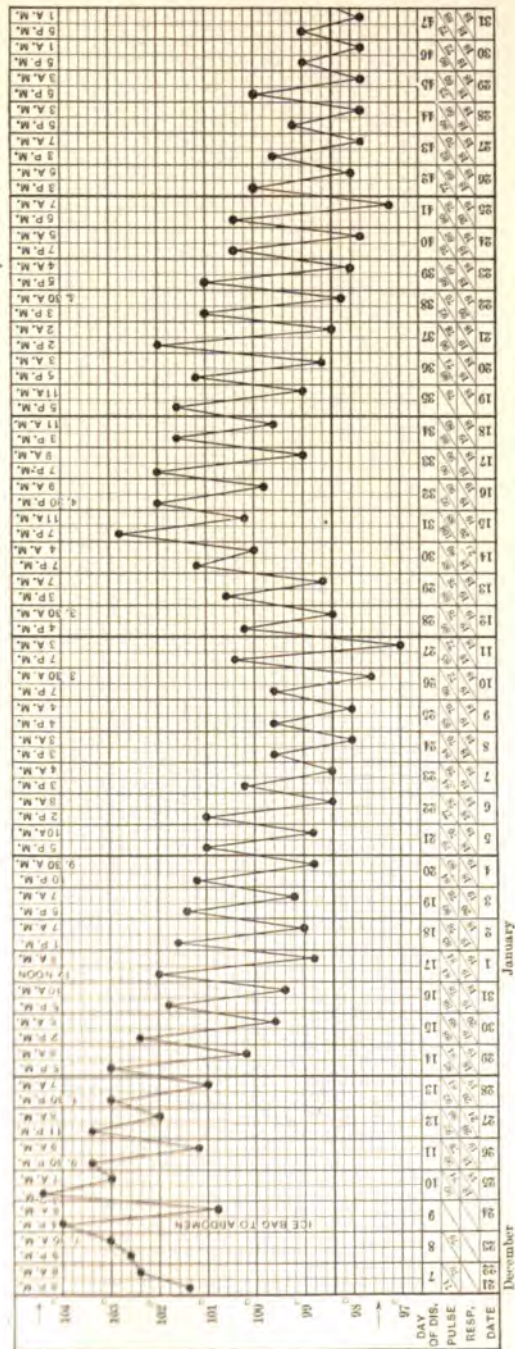
The *temperature curve* shows a gradual ascent during the first four or five days of the disease, with morning remissions. The temperature rises a degree or two in the evening and falls half a degree or a degree in the morning. This step-ladder ascent is characteristic. By the end of a week a temperature of 103°, 104°, or 105° F. is reached, and it remains continuously high, with slight morning remissions, during the second, and less frequently during the third week. In the third or fourth week the morning fall of temperature gradually becomes greater, and by the end of the week the temperature sinks below the normal in the morning, often with marked oscillation the last week. Variations in this typical fever curve are seen when there is a sharp rise at the onset or a pseudocrisis at the termination of the disease. The fever may be very moderate, remittent, or inverse in type. Sudden falls during the course of the disease usually indicates intestinal hemorrhage. Return of the fever after several days of apyrexia, indicates either a reinfection (relapse), a late complication, or may be due merely to extraneous factors, as worry, constipation, etc., acting upon the irritable thermogenic centre. The *pulse* is full, and in favorable cases slower than the pyrexia would lead one to expect. In the second week it is markedly dicrotic. The *heart-sounds* are unchanged apart from complications, but in the second and third weeks the first sounds often are feeble. The *respiration* in uncomplicated cases increases in frequency with the rise of temperature. It usually ranges between 24 and 36. The *nervous symptoms* are often very prominent. In mild cases they consist of hebetude and nocturnal delirium, or they may be absent altogether. Usually, however, by the beginning of the second week there is some mental confusion, with nocturnal delirium. In more severe cases, and later in the disease, the delirium is of a low muttering character, with more or less continuous hallucinations of sight and sound. The patient can be aroused by a question and makes an intelligent answer, but speedily lapses into semiconsciousness. Picking at the bedclothes or efforts to catch imaginary objects are very common. Sometimes the delirium is wild and noisy, rarely the delirium has been so active as to simulate acute mania. Stupor may alternate with delirium. Rarely the patient lies with wide-open eyes, apparently staring fixedly at some object, but really unconscious (coma-vigil).

In severe cases the patient has marked twitching of the tendons, and jactitation. He is wakeful and restless, wearing himself out. The hands and lips tremble, and he keeps muttering to himself all the time.

Convulsions are rare, but may occur in children. Sometimes there are considerable hyperesthesia and tenderness along the spine. On the seventh or eighth day the *eruption* appears. It consists of small, ve slightly elevated, rose-colored papules, which disappear upon pressu and come out in successive crops, each papule lasting three or four d The spots are most common over the abdomen and back, but

THE SPECIFIC INFECTIOUS DISEASES

FIG. 168



Typical chart, mild typhoid fever with an intercurrent relapse. (Original.)

occasionally found elsewhere. They are usually few in number, a half-dozen or dozen.

During the latter part of the second week, and throughout the third week, the symptoms are apt to be intensified. The temperature remains high or even reaches a higher point. Delirium is more decided and constant. The heart grows weak and the pulse increases in frequency. Some degree of hypostatic congestion of the lungs is usual. Diarrhea may be troublesome; intestinal hemorrhages may occur. Tympanites may become so great as to interfere with respiration and circulation. The tongue is dry, brown, sometimes glazed and fissured, and sordes often collect on the teeth. In cases ending in recovery, 90 to 93 per cent., the temperature begins to fall in the mornings; and the severe symptoms gradually abate. It may be interrupted by a relapse, in which the original symptoms are reproduced, with high temperature, but the duration is shorter.

Varieties.—It is now well known, as Osler forcibly states, "that typhoid fever is no more primarily intestinal than is smallpox primarily a cutaneous disease." Thus there may be manifested (1) typhoid fever with intestinal symptoms, as described above; (2) typhoid fever with general infection or typhoid septicemia, in which the symptoms are entirely those of an infection without intestinal manifestations; (3) typhoid fever with more intense symptoms from other organs than the intestines. The lungs, the spleen, the kidneys, and the cerebrospinal meninges are the structures invaded.

Varieties are also based upon the severity of the disease; hence we have the abortive, ambulatory, and grave forms.

The *abortive* form is so named because of the abbreviated course of the disease. The symptoms are sufficiently well-marked to make the diagnosis clear, but the type is mild, and in a week or two convalescence is established.

In the *ambulatory* form, commonly called walking typhoid, the symptoms are so slight that the patient persists in his daily occupation until the disease is disclosed by chance or by the accident of perforation, hemorrhage, or sudden delirium.

Grave forms are due to special severity of some symptoms or group of symptoms, such as hyperpyrexia; toxemia, profound stupor, coma, or intense nervous phenomena; inability to take or retain sufficient nourishment; profuse diarrhea and intestinal hemorrhage; great adynamia with weak heart and a tendency to cyanosis. In other cases the gravity of the disease results from the existence of complications.

Complications and Sequelæ.—Typhoid fever may be accompanied by a large number of complications.

1. **GASTRO-INTESTINAL COMPLICATIONS.**—The most important complications of typhoid fever are intestinal hemorrhage and perforation. *Hemorrhage* is recognized by a sudden fall in temperature, increased rapidity of the pulse, and signs of shock associated with passage of black or tarry stools. It occurs usually between the se-

and fourth week. Small hemorrhages may be manifested only by slight discoloration or blood-tinged stools. *Perforation* is most common during the third week. It is diagnosticated by the occurrence of sudden sharp abdominal pain, associated with tenderness, rapidly developing rigidity, a leukocytosis and elevation of the blood-pressure. At times symptoms of collapse, coincidently appear. In a short time the findings are those of a diffuse general peritonitis.

Acute cholecystitis, recognized by pain, tenderness, and rigidity in the gall-bladder region, is a frequent but somewhat less common complication. *Cholangitis* and *obstructive jaundice* are rare complications. *Ulcerative stomatitis*, *parotitis*, and *rupture of the spleen* are other possible complications of this tract.

2. RESPIRATORY COMPLICATIONS.—*Bronchopneumonia* or *hypostatic congestion* is a frequent and grave complication in the last weeks of a severe attack. *Lobar pneumonia*, *pleuritis*, or *laryngeal ulceration* are occasional complications. An *initial bronchitis* is practically always found.

3. CIRCULATORY COMPLICATION.—Cloudy swelling of the myocardium is a usual complication in cases associated with high fever. *Endocarditis* and *pericarditis* are rare. Thrombosis of the left femoral vein, or more rarely the right, occurs in about 3 per cent. of all cases. It is recognized by a rise in temperature, tenderness and pain along the course of the vein, and edema of the affected leg. A rare complication is occlusion of the femoral artery by a thrombus.

4. NERVOUS COMPLICATIONS.—*Neuritis* is an occasional complication usually of one nerve, as in "tender toes," although occasionally multiple. *Meningitis* is extremely rare, as are other possible nervous complications, *e. g.*, epilepsy.

5. MISCELLANEOUS COMPLICATIONS.—*Periostitis* and *osteomyelitis* are quite frequent and persistent complications, as is *otitis media*, often causing *mastoiditis*. More rare in occurrence are *pyelitis*, *cystitis*, and *acute nephritis*. Ocular complications are exceedingly uncommon. *Bed-sores* and *furunculosis* are relatively common in severe cases in spite of careful nursing.

SEQUELÆ.—As a sequel of typhoid fever, gall-stones are remarkably frequent. In addition, the typhoid bacilli may persist for years in the gall-bladder of the so-called carriers to be periodically discharged, each time acting as a source of possible infection.

Post-typhoidal psychoses develop at times in those who have had marked cerebral symptoms during the course of the disease. The "typhoid spine" makes its appearance soon after the patient starts to get around. It may possibly be a simple neurosis, but more probably is an actual spondylitis or perispondylitis.

Laboratory Diagnosis.—The blood characteristically shows a leukopenia. The differential count usually demonstrates a relative increase in the mononuclear cells. A polynucleophilic leukocytosis or a marked increase in the polynuclear denotes a secondary infection. A bacillemia

is always present, and during the first week of the disease the recovery of the typhoid bacillus by blood culture is possible in a large proportion of cases. After the first week positive results are not so frequent and in addition not so necessary, as the specific agglutination test of Widal can usually be obtained by the end of the sixth or seventh day, persisting for months. A more rapid and easily applied agglutination test than the Widal reaction has been suggested by Bass and Watkins.

The urine is scanty and high-colored, acid, and contains traces of albumin and casts until the temperature starts to descend.

The diazo reaction is usually present. The *typhoid bacilli* are found in about one-third of all cases in the urine by cultural methods frequently before the Widal reaction is positive. (For methods of performing the various diagnostic tests see Section V, Chapters XXIV and XXVII.)

Austrian has devised an anaphylactic skin test which is said to be positive in a large number of cases. The injection immediately under the skin of dead typhoid bacilli of many strains, causes a more or less pronounced cutaneous reaction at the site of inoculation in patients sick with typhoid fever.

Diagnosis.—A typical case of typhoid fever ought not to be mistaken for any other infection, but atypical cases are numerous. In the first week the epistaxis, bronchitis, leukopenia, and diarrhea are the main diagnostic points, in the second week, the rose spots, enlarged spleen, temperature curve, pulse-rate, and Widal reaction are the major criteria.

Typhoid fever not only simulates, but is simulated by other diseases. For example, examination of the blood is at times necessary to exclude the *estivo-autumnal* type of malaria, which often is simulated by typhoid fever. (See page 526.)

Appendicitis is more likely to be mistaken for typhoid fever than the converse. In appendicitis the onset is more abrupt and the local symptoms are more pronounced than in typhoid. Pain and tenderness are prominent in appendicitis, and more acute in the right iliac region, and the attack is often introduced by chilliness and vomiting. Headache is not a prominent symptom, while bronchitis and enlargement of the spleen are absent. Leukocytosis is a valuable sign.

Simple continued fever or fever of undetermined cause is distinguished from typhoid fever of a mild type, principally by the absence of bronchitis, of enlargement of the spleen, of epistaxis, and of the characteristic eruption of typhoid fever.

Typhus fever is distinguished by its sudden onset, the bespotted appearance of the face, with reddened eyelids and small pupils, the absence of abdominal symptoms, and the appearance on the fourth day of macules, which are subsequently converted into petechiae. The mild type of typhus described by Brill is frequently only differentiated by the absence of the Widal reaction.

Relapsing fever differs from typhoid fever in its sudden onset with chill, pain in the epigastrium, but absence of abdominal symptoms and eruption; the conclusive test is the finding of the spirilla in the blood.

Acute miliary tuberculosis at times closely resembles typhoid fever. In both the onset is gradual, with cough and fever. In the former, however, the bronchial symptoms are more prominent, there are apt to be recurring chills and sweats, the temperature is remittent and irregular, emaciation is rapid, and constipation instead of diarrhea is the rule.

In *peritoneal tuberculosis* there is persistent, diffuse pain in the abdomen, and the belly is swollen. The temperature is irregular and may be below normal; nervous symptoms comparable to those of typhoid are wanting.

Cerebrospinal meningitis and typhoid fever are often extremely difficult to differentiate in the first week. Lumbar puncture should be performed if there is any doubt, not only from the diagnostic but also from the therapeutic standpoint.

In *tuberculous meningitis*, the knee-jerk, and other reflexes are variable, irregularly absent or present. In typhoid fever they are always present. In the former, choroidal tubercles may be seen with the ophthalmoscope.

Ulcerative endocarditis and *septic conditions* generally may at times be confused with typhoid fever, but the laboratory diagnosis, leukocytosis in the first conditions, leukopenia, and the Widal reaction in typhoid, will successfully aid in differentiating the several conditions.

Typhoid fever must not be confounded with *trichinosis*; the peculiar muscular pain and edema do not occur in the former. *Acute nephritis* may simulate typhoid fever, but the character of the urine and the absence of the specific typhoid symptoms, render the diagnosis easy in most cases.

Uremia of a chronic form may simulate typhoid fever, but the character of the urine, the cardiovascular symptoms, and the absence of the Widal reaction suffice to differentiate the two conditions.

Influenza is frequently mistaken for typhoid fever, but the absence of rose spots and the more rapid pulse, as well as a negative agglutination reaction, are sufficient points upon which to base a diagnosis.

The presence of all the physical signs of *hypostatic congestion* or *bronchopneumonia* in the aged often results in the overlooking of a typhoid infection. *Lobar pneumonia*, at those times when it coincides with the onset of the disease, may also cause a similar error.

Paratyphoid Fever.—This infection is due to a bacillus which is closely related to the typhoid and the colon bacillus.

The symptoms are like those of typhoid fever in the larger number of cases. In another, but smaller group of cases, the symptoms are those of gastro-enteritis with fever. The first form corresponds closely to typhoid fever in all its incidents, including the etiology and the occurrence of relapses. It is usually of shorter duration, premonitory symptoms are absent, prostration is early, myalgia is more marked, and the temperature rises more rapidly. In the gastro-intestinal form the temperature rises rapidly after a chill; diarrhea supervenes at once;

vomiting and epigastric pain are marked; while prostration is the dominant feature. The diagnosis can only be established by the serum test. Cultures from the blood or urine would prove positively the presence of this infection.

In using the serum test, both species of the paratyphoid bacilli must be employed, and an agglutination should not be diagnosticated as positive except with high dilution. The blood serum of these cases fails to agglutinate the typhoid bacillus or agglutinates it only in very low dilutions.

Yellow Fever.—An acute, contagious disease, endemic and epidemic on the tropical and subtropical shores of the Atlantic Ocean, characterized by a sudden onset, a duration of a week or less, a characteristic facies, slow pulse-rate, albuminuria, jaundice, and vomiting, with a tendency to hemorrhages.

The specific organism causing yellow fever is as yet unknown, but it is known that it is transmitted by the bite of the mosquito, *Stegomyia fasciata*. Prophylactic measures depend upon the destruction and prevention of growth of these mosquitoes. The disease becomes epidemic in the hot season and disappears upon the appearance of frost, and the consequent disappearance of the mosquito.

Symptoms.—The period of *incubation* varies from a few hours to two weeks, exceptionally beyond the seventh day. The course of the disease may be divided into two stages, the stage of invasion and the stage of collapse, with usually a distinct period of intermission.

First Stage.—The *invasion* is abrupt, and occurs usually in the night. It is marked by chilliness oftener than by a decided chill. The temperature rises rapidly to 102° to 103° or 104° F. The *pulse* is correspondingly increased in frequency at first, but very commonly begins to fall before the temperature, so that later the pulse is relatively slow. The *face* is peculiar and characteristic—it is flushed and somewhat swollen; the edges of the eyelids are reddened; the eyes are watery, glistening, and slightly, but distinctly, tinged with *yellow*; the pupil is small and brilliant.

The *tongue* is large, moist, and coated with white fur. The stomach is irritable and the epigastrium tender. Nausea with repeated vomiting occurs. The fluid is at first of a light greenish yellow, subsequently becoming decidedly bile-stained. The bowels are constipated.

The *urine* almost invariably contains albumin at some time during the first three days. The albuminuria may last only a short time and may be found only in the evening.

During this febrile period the patient complains of headache, pain in the back and limbs, and intense thirst; the mind, however, is usually perfectly clear.

Period of Intermission.—In from two to five days the temperature falls to or below normal, remaining there for forty-eight to seventy-four hours. In more severe cases the period of remission or stage of *collapse* is followed in a few hours by a return of symptoms.

Second Stage.—The jaundice deepens, vomiting becomes more urgent, and in adults is accompanied by much retching. The vomitus is bilious, streaked with blood, or thick and wholly black (black vomit); the temperature may equal or surpass that of the original paroxysm, or it may remain depressed.

In any event the pulse is apt to be slow, often from 40 to 60. The urine contains albumin, blood and casts, and there may be suppression, adding uremia to the other toxemia. Convulsions at this stage are usually uremic. Hemorrhages may occur from any mucous surface. The gums are tender, swollen, and bleed easily. Death may take place in coma or convulsions.

As in scarlet fever, the patient may be smitten down and die in a few hours from the time he was in apparent health. In other grave cases the temperature remains high, and rises instead of falls on the third or fourth day. The *duration* of the disease is from two to five or six days; if a typhoid state develops, it may last ten days to two weeks.

Complications are not common. Second attacks are extremely uncommon.

Laboratory Diagnosis.—The urinary findings already mentioned are the significant laboratory aids in diagnosis.

Diagnosis.—Yellow fever is distinguished from *malarial fever* by the slow pulse, the characteristic facies, the early transient albuminuria, the early and deep jaundice, and the absence of enlarged spleen. *Dengue* (see page 475).

Malta Fever.—An acute infectious disease, caused by the *Micrococcus melitensis* and characterized by gradual onset and by repeated remissions of the fever. The disease prevails in countries in which the milk of goats is used and by which the disease is transmitted. It occurs occasionally in Texas and the Southwestern States of this country. The alternating febrile and afebrile periods which characterize the disease continue from two months to two years. These periods of fever last from one to three weeks, followed by an apyretic period or a period of abatement lasting from two to ten days. The daily temperature range may be intermittent or remittent, possessing a peculiar wave-like or undulatory character. The patients grow more and more prostrated, become anemic, and usually suffer from constipation. Profuse sweats attend the decline of the daily range, and in many instances the spleen is enlarged. Neuralgias occur in various parts of the body; the joints become enlarged, and fibrous tissues may be the seat of inflammation.

Laboratory Diagnosis.—The organism can be isolated by blood-culture in most cases. A specific agglutination reaction is obtainable. The leukocytes are not increased, although there is a relative increase of the large mononuclears.

Diagnosis.—In doubtful cases suggesting typhoid or malarial fever the several laboratory tests will disclose the real nature of the disease.

Erysipelas.—An acute, slightly contagious disease, caused by the *Streptococcus erysipellatus*, characterized by a sudden onset, with a bright red eruption which, usually in the cases seen by medical men, begins on the face near the nose or mouth and spreads over the entire face and scalp. The infection is also apt to attack persons with open wounds (surgical erysipelas) and puerperal women. One attack predisposes to another.

Symptoms.—The period of incubation is usually from three days to a week. The invasion is sudden and is marked by chill. The temperature rises to 104° or 105° F., and in the next two or three days may rise still higher, continuing for five to seven days, and, as a rule, terminating by crisis. Coincidentally with the rise in temperature the portion of the skin to be affected burns, tingles, is tender to the touch, and may be reddened. The redness increases in intensity and extent, while the skin is swollen and slightly edematous. Vesicles and blebs often form when the inflammation is very intense. The redness disappears upon pressure, but quickly returns; sometimes it has a dusky, purplish hue. The redness and swelling begin to subside at the periphery of the part first attacked. The part of the face first affected is usually the nose, spreading from there to the cheeks, eyes, forehead, and at times to the scalp, ears, and less frequently to the neck. In pronounced cases the swollen face and the closed eyes, the thickened lips, and enlarged nose cause a great distortion of the facial features. The advancing margin is raised, tense, and brawny; the line is thus sharply drawn between healthy and inflamed tissue. The submaxillary glands are more or less enlarged. On the body the eruption spreads over a greater extent than when primary on the face, hence its name, the "red runner" (erysipelas migrans). While the eruption is extending, the fever continues and is sometimes alarmingly high. The pulse is frequent and soft.

Complications and Sequelæ.—Pneumonia and nephritis are the most frequent complications. Meningitis, pericarditis, and endocarditis also occur. Erysipelas may extend inward and involve the sinuses, pharynx, and larynx, producing edema and death from suffocation. If the scalp has been involved, the hair falls out. The cervical adenitis may result in abscess; chronic nephritis may develop. Otitis media occurs occasionally, and so do keratitis and abscess of the eyelids. On the other hand, erysipelas is credited with causing the disappearance of lupus and chronic eczema.

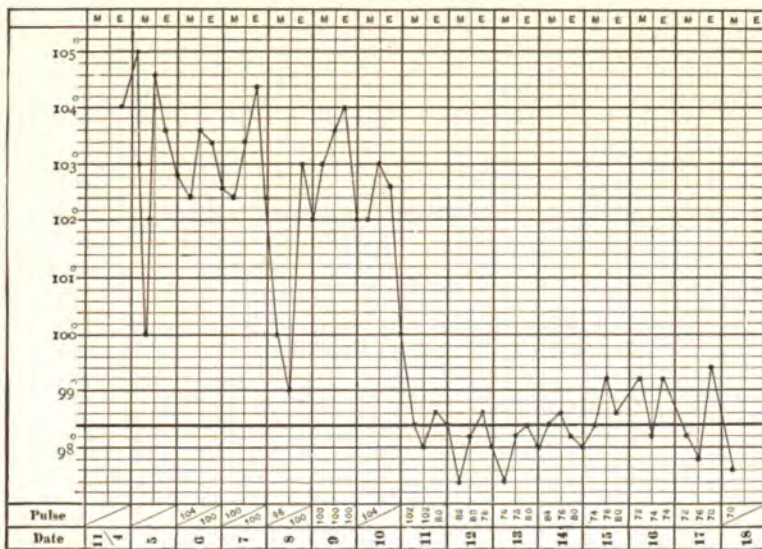
Laboratory Diagnosis.—The neutrophilic hyperleukocytosis that is present is usually in direct relation to the severity of the local condition. In rare cases, the streptococci can be isolated by blood culture. Urine is febrile in character unless acute nephritis complicates the case, when the urinary findings are those of that disorder.

Diagnosis.—The local lesions associated with high fever are usually sufficiently characteristic to make the diagnosis. Acute ecthyma, malignant pustule, dermatitis, and erythema may at times cause confusion in the diagnosis.

Lobar Pneumonia.—Acute pneumonia, croupous or lobar pneumonia, is an infectious inflammatory disease excited by the *Micrococcus lanceolatus* (*Diplococcus pneumoniae*, *pneumococcus*) involving the vesicular structure of the lungs, characterized by sudden onset of fever, severe constitutional as well as respiratory symptoms, and terminating by crisis.

Symptoms.—*Mode of Onset.*—The invasion of pneumonia is usually sudden, and is marked by a *chill*. The temperature rises rapidly, and may reach 104° or 105° F. in the first twelve hours after the chill. With the fever, the patient complains of severe headache and *pain* in the side, and has a short, quick cough and sometimes vomits. The pulse is moderately accelerated and the respiration soon becomes very frequent.

FIG. 169



Pneumonia; sudden rise; termination by crisis; pseudocrisis on eighth day. (Original.)

The face is apt to be flushed, and there may be a circumscribed red spot on the cheek. In some cases the course resembles that of an infection in which the pulmonary disease is a local manifestation. In such cases there may be prodromata, consisting of headache, general malaise, a slight bronchitis, and digestive disturbances. Then follows the chill.

Later Stages.—At the end of forty-eight hours, or, at the most, of four days, the patient is found lying in bed in the dorsal position, or on the affected side. The face is flushed and countenance anxious, the respiration hurried, the *ala nasi* play vigorously. The temperature varies little from the first day's rise, the chest pain has subsided, and the short, dry cough is now attended by viscid expectoration. The respiration continues hurried, the pulse full and bounding.

Duration and Course.—The duration of the disease is from one to two weeks. It may subside by crisis on the third, fifth, seventh, or ninth day, or gradually by lysis. Crisis is marked by a profuse sweat, a copious discharge of limpid urine, or sometimes by a few loose movements of the bowels, accompanying a fall of temperature to or below normal, and a marked amelioration of symptoms. The following symptoms require more detailed consideration.

Respiratory Symptoms.—Chest pain, cough, hurried respiration of a peculiar type, and expectoration are characteristic. The *chest pain* (from involvement of pleura) is sharp, stabbing, or lancinating, and increased by breathing. Its seat always indicates the side affected. *Cough* is short and dry, smothered and painful; it soon becomes softer and painless as the expectoration becomes free. The cough is followed by *expectoration*, which is at first viscid mucus, but gradually becomes reddish brown from admixture of blood—*rusty sputum*. This sputum is characteristic, almost pathognomonic. It is expelled with difficulty from the mouth, being thick, tenacious, and homogeneous. Characteristic symptoms of pneumonia are the increased frequency and the type of the *respiration*. The rate reaches 40, 50, or even 60 per minute. The pulse, on the contrary, does not increase in frequency in the same proportion; hence the normal ratio of respiration to pulse of 1 to 4 becomes 1 to 3 or 1 to 2. Inspiration is short, expiration quick and often attended by an expiratory noise or grunt. The long pause may take place after inspiration instead of expiration.

Fever.—In twelve hours after the chill the temperature reaches 104° to 105° F. It remains at this point, obeying the laws of diurnal variation. At the end of the third, or more frequently the fifth, seventh, or ninth day, *crisis* takes place; the fall is abrupt, and the normal or a subnormal temperature may be reached in from five to fifteen hours. *Pseudocrisis*, as the accompanying chart indicates, may precede the true crisis by twenty-four or forty-eight hours. The decline, however, may take place by lysis. Protracted fever indicates delayed resolution or the occurrence of a complication.

Cerebral Symptoms.—In some cases, especially in children, the onset of the disease may be marked by a *convulsion*. This is said to occur more frequently in apical pneumonias than in the pneumonias of the base. Headache and delirium are so pronounced in some cases as to simulate meningitis. *Delirium* may occur during the height of the fever, and occasionally is maniacal. Nocturnal delirium may be a constant symptom in very grave cases. In the later stages of grave or fatal cases a low form of delirium, with a tendency to coma, is common.

Heart and Pulse.—The pulse is small at the time of the chill, but becomes full and bounding as the fever persists. In healthy adults it is rarely over 110. In the debilitated it may be very frequent, small, and feeble; in the aged, frequent and dicrotic. The blood-pressure and pulse-pressure usually preserve their normal state. A valuable

prognostic sign is the relation of blood-pressure to pulse-rate. If the blood-pressure falls below the pulse-rate the prognosis is grave, and the converse is true. The heart-sounds are clear. The left ventricle acts forcibly. The pulmonary second sound is accentuated. If dilatation and failure of the right ventricle take place, the area of dulness may extend beyond the right edge of the sternum, an epigastric impulse be noted, and turgescence of the veins in the neck and cyanosis becomes marked, but above all, the previously accentuated pulmonic second-sound may become weak or disappear.

Gastro-intestinal Symptoms.—Vomiting frequently occurs in children at the onset, and in adults at times. The appetite is lost; the tongue is furred, and may become dry and brown; the bowels are constipated; tympanites is often present; epigastric tenderness is frequently observed in children; jaundice occurs in a certain number of cases; and the *spleen* is enlarged.

Cutaneous Symptoms.—Herpes on the lips, the nose, or the genitals is of common occurrence. Sweating occurs with the crisis, or if heart failure is imminent.

PHYSICAL SIGNS.—*Solidification.*—This consists in a diminution of the amount of air and an increase of solid contents. On *inspection* is found diminished movement. On *palpation* the results of inspection are confirmed and increased vocal fremitus discovered; *percussion* in the first stage gives impaired resonance or Skodaic resonance; in the stage of hepatization, dulness or flatness, but without any wooden quality or marked resistance. *Auscultation:* in the early stage, that of congestion, the respiratory murmur is suppressed and crepitant rales are heard at the end of inspiration. On full inspiration or after cough a bronchovesicular respiration is brought out. When consolidation has taken place, the respiratory murmur is bronchial. Rales, if present, are moist subcrepitant rales due to associated bronchitis, or a few crepitant rales may persist, and a friction-sound be heard. When resolution sets in, the crepitant rale reappears quickly followed by moist subcrepitant rales, heard both on inspiration and expiration, while dulness gradually yields to impaired resonance. The respiration loses its bronchial character and again acquires a vesicular element before becoming completely normal. It may be a week or two, before the percussion-note becomes perfectly clear and rales wholly disappear. When there is *delayed* resolution the physical signs of solidification persist for a longer or shorter time, often for weeks. There may be a complete cessation of fever and other symptoms or more commonly the temperature defervescence is by lysis, becoming intermittent or remittent; the pulse rate remains rapid, and there is some cough without expectoration. Wasting may be marked.

The physical signs are modified by the intensity of the inflammation in the lung structure and by the pleural complications. In *massive pneumonia*, resulting in occlusion of the bronchi, for instance, the auscultatory signs are absent. On percussion, the lung is absolutely

flat. There is no fremitus or tubular breathing. The physical signs resemble those of pleurisy with effusion. In *double pneumonia* the physical signs are bilateral. *Apical pneumonia* presents the signs of solidification at the apex and the symptoms usually of a more severe infection than when the lesion is limited to the base of the lungs. In *central pneumonia* the physical signs may be delayed until the third or fourth day. A few rales or feeble breath-sounds over a small area may be the only indication of a possible lung process. In the *aged* the physical signs are obscure. In patients with laryngeal disease or marked obstruction in the nasopharynx the physical signs may be indefinite. Bronchial breathing may not be heard unless the patient takes a full breath or coughs. In this class of cases, as well as in those with feeble respiratory movement, as the aged, the weak, and in those suffering from some other disease, as tuberculosis, the physical signs are not made out because of the deficiency of respiratory movements.

Varieties.—*Migratory Pneumonia*.—Pneumonia of this variety is characterized by invasion of different parts of the lungs at different times, the hepatization resolving in one part while it spreads to another.

Abortive pneumonia is characterized by a short course of from one to three days and incomplete solidification of the lung.

Terminal Pneumonia.—In patients with chronic diseases pneumonia may insidiously develop without much change in the fever and pulse-rate and may not be suspected until death approaches or the condition is found at autopsy.

Asthenic pneumonia occurs in those much exhausted, in depraved health, or exposed to unhygienic surroundings. It is found also in a mixed infection, in Bright's disease, and in drunkards. The characteristic features of this form of pneumonia are the great physical prostration, the weak heart action, and the scarcity of physical signs. The fever is low, the respiration and pulse frequent, and vomiting and delirium, as well as all nervous symptoms, are more common than in the ordinary form. Cough and expectoration are frequently absent or the latter may be either bloody or resemble prune juice. The physical signs are not definite. The disease may rapidly prove fatal or may linger for a long time, the patient only gradually coming out of a low typhoid state. It is always dangerous.

Pneumonia of this type is probably the result of the overwhelming general action of the toxins of pneumococci circulating in the blood (septicemia) rather than their local action.

Pneumonia in infants is characterized by nervous symptoms. Repeated convulsions and active delirium may be most pronounced, followed by torpor and coma. There is no sputum and but little cough. The apex of the lung is usually affected.

Pneumonia in the aged is characterized by latency of symptoms. There is but little cough and expectoration. A tendency to the typhoid state, however, is pronounced. The physical signs are obscure.

Pneumonia in alcoholic subjects also develops insidiously and may be masked by the symptoms of delirium tremens. The temperature may be the only indication of infection, as there is neither pain, cough, expectoration, nor dyspnea.

Pneumonia with Other Infections.—*Staphylococcus* and *Streptococcus pyogenes*, the colon bacillus, and *Bacillus pneumoniae* (Friedländer) are often found with the pneumococcus, and may predominate, inducing a *mixed infection*. The microorganisms which cause diphtheria, typhoid fever, influenza, and the plague, may cause a condition which resembles that of lobar pneumonia in the extent of the solidification.

Complication.—*Relapses* are exceedingly rare, although recurrences are common. *Pleuritis* occurs in all cases in which the inflammatory process involves the periphery of the lung.

Pleural effusion is found in 6.26 per cent. (Osler's *System*, Musser, and Norris) of all cases. It is usually not recognized until the subsidence of the pneumonia.

Empyema develops in about 2.2 per cent. of the cases. An intermittent fever associated with a leukocytosis, a small area of dulness, and absent fremitus and voice-sounds are symptoms so suggestive that the exploratory needle is indicated.

Abscess or gangrene of the lung are occasional terminations of a pneumonia, recognized by persistence of fever which becomes septic in type as well as by purulent expectoration in the first condition, and the latter by the fetid sweetish odor of the breath.

Acute endocarditis or *pericarditis* are relatively frequent major complications, as is acute nephritis, while meningitis and acute dilatation of the stomach are rare complications. Of the less dangerous or minor complications may be mentioned jaundice, arthritis, tonsillitis, parotitis, phlebitis, and otitis media.

Laboratory Diagnosis.—The characteristic sputum of pneumonia has been discussed. The recognition of the pneumococcus in stained specimens, unless in very large numbers, is of little value, as they are normally found so frequently in the mouth.

The Blood.—The leukocytes are regularly increased, averaging about 20,000, unless resistance is very poor. The polynuclear neutrophiles are uniformly increased. With appropriate methods the pneumococcus can be isolated by blood culture in a large proportion of cases.

The *urine* presents the characteristics of febrile urine. Albumin is found in over half of all cases of lobar pneumonia; hyaline casts usually occur simultaneously. A diagnostic factor of importance is the diminution or absence of chlorides in the urine.

Diagnosis.—The diagnosis is based upon the aggregation of special symptoms. The mode of onset, the chill, the course of the fever, the pain in the chest, the cough, the peculiar expectoration, the dyspnea, the abnormal pulse-respiration ratio, the peculiar character of breathing, the physical signs, and leukocytosis are the phenomena of the symptom-complex. It must be remembered that in children, in the aged, in drunk-

ards, in cases of chronic disease, the type is different. In all of the above cases, if there is *fever* without cause, although no pulmonary symptoms are present, the lungs must be examined repeatedly. In many such cases the physical signs are obscured because respiratory action is enfeebled by the primary condition.

Pneumonia must be distinguished from other acute inflammatory affections of the lung and pleura and from acute pneumonic phthisis. The evidence for each is considered in the respective sections. The presence of leukocytosis serves to distinguish it from acute tuberculosis and from typhoid fever, meningitis, and influenza. To distinguish pneumonia from pleurisy with effusion, the aspirator may be used.

Occasionally pneumonia at the onset may simulate acute abdominal conditions as appendicitis or peritonitis but careful physical examination will disclose the true lesion.

Tuberculosis.—An infectious disease, the course of which may be acute or chronic, caused by the *Bacillus tuberculosis*. Invasion of the body by the microorganism may give rise to general infection, with an eruption of miliary tubercles in most of the organs and structures of the body, or to a local infection of one organ. General tuberculosis is acute; local tuberculosis may be acute or chronic. General tuberculosis is usually secondary to a focus of local infection. In acute tuberculosis the serous membranes, the lungs, liver, kidneys, lymphatic glands and spleen, the bone-marrow, and choroid coat of the eye may be invaded in whole or in part. In chronic tuberculosis the lymph glands, the lungs, the serous membranes, the tissues and organs of the alimentary canal, the liver, the organs of the genito-urinary system, or the brain and cord are individually invaded.

Tuberculosis may occur at any age. Lymphatic, joint, and meningeal tuberculosis is most common in the first decade of life; the mesenteric glands are particularly subject to invasion at this period.

Certain inherited anatomical characteristics, for example, the phthisical chest, predispose to tuberculosis. More important, however, is an unhygienic manner of living or working.

Diagnosis.—The diagnosis of any form of tuberculosis is aided by (1) the determination of a source of infection, when this is possible; (2) the symptomatology of the general or local infection; (3) the bacteriological examinations; (4) certain specific tuberculin reactions.

1. The infections may take place through (a) inhalation; (b) alimentation (rare) or (c) direct inoculation (very rare) of the tubercle bacilli, so that at times the source of the infection may be directly traced to a member of the family, to an infected milk-supply, or to an autopsy wound.

2. The symptomatology of tuberculosis of the various organs is discussed in the chapters devoted to disease of special organs.

3. The discovery of the *Bacillus tuberculosis* in any inflammatory area, or any product of inflammation, as serum, blood, pus, or the secretion from any gland or mucous membrane invaded by the disease, establishes

at once the diagnosis of this condition. The method of determining the presence of this microörganism is fully detailed elsewhere.

4. The specific tuberculin tests include the subcutaneous injection of tuberculin, the cutaneous reaction of von Pirquet and Morro, and the conjunctival reaction of Calmette.

The Tuberculin Tests.—The subcutaneous injection of tuberculin in tuberculous subjects causes a definite phenomena or reaction.

Phenomena of Reaction.—About twelve hours after the injection of tuberculin the temperature rises rapidly. In the course of a few hours it has risen two or three degrees, is attended by malaise, pain in the head, back, and legs, and sometimes nausea or vomiting. The maximum temperature is maintained for two or three hours, and then a gradual decline to the normal takes place. The normal temperature is reached in from twenty-four to thirty-six hours. The whole period of the reaction, from the time of the injection until the termination of the fever, is about forty-eight hours. With the fall of temperature to normal the constitutional symptoms subside. The accompanying chart (Fig. 170) shows the course of the fever in a typical reaction.

FIG. 170



Typical reaction with tuberculin. (Original.)

Method.—For twenty-four to forty-eight hours preceding the test the patient's temperature should be taken every two hours to determine the range at this period of the disease. The injection should be made at bedtime or in the morning.

The site of the injection is usually the interscapular space. The initial dose should never exceed 5 mg., and if negative an increasing quantity, injected every second or third day up to 10 mg. For children $\frac{1}{20}$ mg. to $\frac{1}{10}$ mg. may be the initial dose.

At the point of injection a little redness and infiltration, with tender-

ness to the touch, are observed. In pulmonary tuberculosis in which physical signs are obscure some auscultatory phenomena which were previously absent may be found during the period of a reaction.

It must be remembered that a negative result with large doses of tuberculin is of more value than a positive one. In the former instance one can affirm that tuberculosis is absent, as well as that there is no old focus in any of the organs. It must also be remembered that the test should only be employed after all other means have failed to make a positive diagnosis.

The cutaneous reaction of von Pirquet is performed as follows: The skin of the upper arm is cleansed with alcohol and ether. Three small spots about an inch apart are then scarified with any instrument, preferably with a dull chisel-shaped scarifier made for the purpose, just deep enough so as not to draw blood. Upon the upper and lower spot (the middle spot being used as a control) is placed a drop of old tuberculin, which is then allowed to dry in the air.

Reaction.—In from twenty-four to forty-eight hours a small red papule appears at the site of scarification which is usually accompanied by some redness in the skin bordering on the papule. The intenseness of the reaction usually is in direct relationship to the activity of the tuberculous infection.

The reaction may be said to be of negative value in adults, as it remains positive in some cases that have at some time or another been infected with tubercle bacilli. A positive reaction is therefore of some value only in conjunction with other signs and findings, but a negative reaction should absolutely exclude tuberculosis except in the advanced stages when the reaction is always negative as a rule. Even in children in whom the reaction is of the greatest value von Pirquet found that 36 per cent. of children over five years of age, clinically non-tuberculous, gave a positive reaction.

The cutaneous reaction of Morro is performed by rubbing 50 per cent. tuberculin ointment into the skin for a minute. The reaction is positive if a crop of small red papules develop at the site of inunction. In a series of von Pirquet and Morro tests, performed by the reviser upon the same patients at the same time, the results of the two tests paralleled each other very closely.

The conjunctival reaction of Calmette is secured by injecting a drop of 1 per cent. tuberculin into the conjunctival sac. The reaction is manifest in at least forty-eight hours by the appearance of palpebral redness and congestion of the whole conjunctiva. The reaction is less sensitive than the others but the ocular complications that have resulted in some cases from its use should mitigate against its general employment.

Acute Miliary Tuberculosis.—This is a manifestation of tuberculosis, secondary to a breaking down of a localized area of tuberculosis somewhere in the body and is analogous to pyemia, occurring as a result of organism from a localized focus of suppuration, entering the blood-

stream with the formation of metastatic abscesses. One of three main types of the disease, according to the predominant manifestations of the symptoms, are usually seen: (1) the typhoid form, (2) the pulmonary form, (3) meningeal form. The last two forms are discussed elsewhere (see page 595 and page 753).

The Typhoid Form.—The onset is usually insidious. The patient gradually becomes weak and feverish. The fever rises more or less gradually, but is quite irregular, with pronounced diurnal variations. At times the inverse type is seen. The pulse is rapid and feeble; the respirations are increased and the patient is usually cyanotic. He gradually becomes stuporous, dull, and profoundly toxic (active delirium is rare), and passes into coma, in which he may die, or pulmonary or meningeal symptoms may develop and apparently cause death.

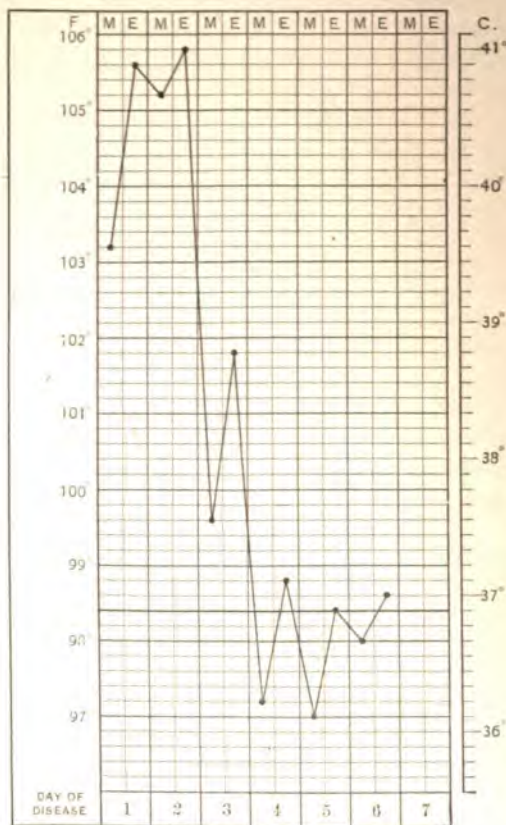
Diagnosis.—The differentiation between typhoid fever and acute miliary tuberculosis is frequently extremely difficult. In the latter condition an enlarged spleen, a macular eruption, an initial bronchitis, constipation, and a diazo-reaction are frequently found. However, in tuberculosis of this type the fever-curve is not so regular as in typhoid, the bronchitis is usually persistent and cyanosis is present and a leukocytosis may be found. The diagnosis must frequently be made only by the presence or the absence of the Widal reaction. The tuberculin reactions are usually valueless.

Influenza.—A somewhat contagious disease, caused by the influenza bacillus of Pfeiffer, occurring in widespread epidemics, having a very short period of incubation, and characterized by great prostration, marked nervous symptoms, and catarrhal inflammation of the respiratory or gastro-intestinal tracts, or both. There is great liability to relapse and to complications, which are generally pulmonary or nervous.

Symptoms.—The disease generally begins with the ordinary symptoms of coryza; but the headache over the eyes and root of the nose is more severe and may be so agonizing as to mask all other symptoms. The lacrymation, rhinitis, and tormenting cough are all usually worse than in ordinary coryza. Physical weakness, weariness, and depression of spirits are almost invariably present, and they sometimes reach an extraordinary degree. Fever is usually moderate (100° to 102° F.), but may be 104° to 105° F. for several days, and then gradually subside. It may terminate by crisis or may assume an intermittent or remittent type. Nausea and vomiting are not uncommon, especially in the morning, at which time also the patient frequently feels worse than he does later in the day. Sleep is broken and restless, and may be accompanied by drenching perspiration. Severe neuralgic pains are common. In some cases the disease attacks the gastro-intestinal tract especially, and diarrhea and vomiting are the prominent symptoms. In other cases the predominant symptoms are nervousness, and great pain with profound prostration mask the catarrhal symptoms. Torpor and delirium may be present.

The duration of the disease is from a few days to a few weeks. Convalescence is remarkably tedious, and is characterized by persistent weakness. Sweats are often annoying during this time. Relapses are common.

FIG. 171



Temperature in influenza—interrupted crisis. (Wilson.)

Complications.—The most frequent complications are those involving the lungs. Intense bronchitis and bronchopneumonia are extremely common. Pleural effusions, empyema, and pulmonary abscess or gangrene are less frequent.

The usual complications of the nervous system are meningitis, brain abscess, encephalitis, and neuritis. The arrhythmia, tachycardia, bradycardia, precordial pain, syncope, or palpitation that frequently occur are probably a cardiac manifestation of extreme toxemia.

Otitis media and conjunctivitis (frequently), iritis, catarrhal jaundice, and nephritis (rarely) may also develop.

Laboratory Diagnosis.—The stained *sputum*, when obtainable, contains the causative bacillus. The total leukocytes are either moderately increased or else they remain normal. The lymphocytes are relatively increased.

Diagnosis.—Influenza in the great majority of cases is easily recognized, particularly if the disease is epidemic. In certain cases, however, it is to be differentiated from *bronchopneumonia*, *typhoid fever*, and *cerebrospinal meningitis*.

Cases in which the disease sets in with high fever and marked chest symptoms are very apt to be mistaken for *bronchopneumonia*; but the headache and prostration are more intense, while the respiration is not so frequent. Physical exploration shows that both lungs are involved, though often not to the same degree. Resonance is impaired, and auscultation shows subcrepitant rales, which seem to be due to an edematous condition of the lung tissue associated with a diffused bronchitis. If diarrhea is one of the symptoms, *typhoid fever* has to be excluded. This is extremely difficult in the first two or three days, but, as a rule, headache, backache, nausea, and sleeplessness, are at this time greater in influenza, and the onset is acute. Influenza can be distinguished from *cerebrospinal meningitis* by noting the fact that it begins with coryza, whereas the latter often sets in with chill, vomiting, and faintness; the headache in the former is usually frontal, in the latter occipital, and accompanied by stiffness of the back of the neck.

Cerebrospinal Meningitis.—An acute, infectious, and mildly contagious disease, caused by the meningococcus (*Diplococcus intracellularis meningitidis*), endemic and epidemic, occurring most commonly in children under fifteen years of age, characterized by intense pain in the back and head, hyperesthesia, retraction of head and neck, delirium, coma, convulsions, and vomiting.

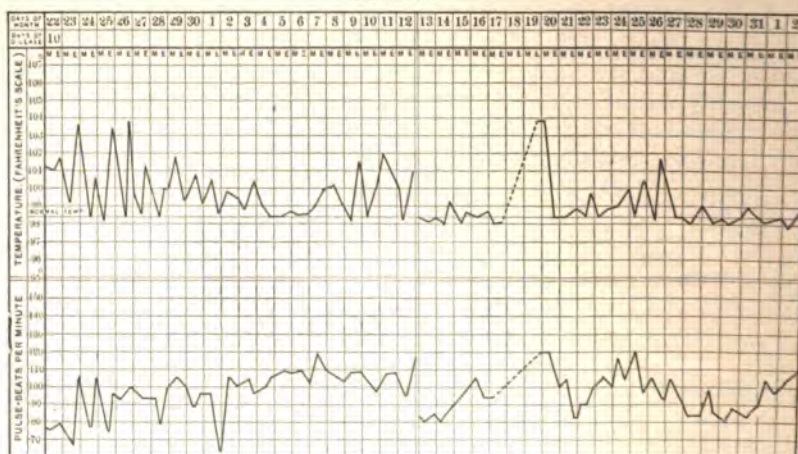
Symptoms.—The period of incubation is unknown, but is probably short. It is free from prodromal symptoms. The invasion of the disease is abrupt, usually with a severe or a convulsive chill. Then quickly follow repeated vomiting, intense headache, sometimes accompanied by backache, retraction of the head, delirium, and extreme prostration.

The extraordinary irregularity of the temperature is most striking. It is often of very short duration, followed by a prolonged subnormal temperature. It may be high from the onset of the disease, or remain below 100° for several days, and then suddenly rise to a great height. Remissions and exacerbations may attend many of the cases. The most marked feature, apart from the irregularity of the temperature, is the inequality between the pulse and the temperature. In some instances the pulse is rapid and the temperature is normal or subnormal, while later in the disease the pulse may be slow when the temperature rises to a considerable height.

The face is pale and livid, expressive of suffering. Simple stiffness of the muscles of the neck may prevail, or the spinal muscles become

rigid and the head is often retracted. Less frequently trismus occurs and the back is arched. Delirium is common at night. It may develop very early or appear at a late period of the disease. It is sometimes violent or low and muttering. Delirium may alternate with tonic or clonic convulsions and with stupor. The pain in the head may be occipital or frontal. The pain in the back becomes more severe, and root-pains dart in all directions. The appetite is poor, the bowels constipated. A remission may occur on the third day, with temporary improvement of the symptoms.

FIG. 172



Cerebrospinal meningitis, showing irregularity of pulse and temperature. (Councilman.)

The *skin eruptions*, which explain the old name "*spotted fever*," are not always present and exhibit no constant character. Herpes and petechia are the most frequent; in other cases the eruption is a purplish mottling, or is macular, or the eruption resembles that of measles. Herpes is most common on the nose and mouth, then on the cheek, forehead, eyes, and ears.

As the attack progresses there may be strabismus, which is usually divergent, inequality of the pupils, nystagmus, ptosis, and optic neuritis. Vertigo, tinnitus, anosmia, and photophobia are common. Facial paralysis, a monoplegia, a hemiplegia, or a paraplegia may occur. The pulse becomes more frequent and the fever continues. In favorable cases the headache and root-pains abate, and delirium and spasms become less frequent. In unfavorable cases the convulsions may become more severe and end in fatal coma, or the patient may sink into a typhoid condition. Coma may come on in the beginning and continue until death.

Kernig's Sign.—(Kernig, 1884; Netter, 1898). This sign is of value only in the diagnosis of meningitis in general. It is determined by flexi-

the thigh on the abdomen until it makes a right angle. When the attempt is made to extend the leg, it will be found that the limb cannot be fully extended if meningitis is present.

OTHER TYPES.—In the *malignant (fulminating)* form of the disease death occurs in a few hours, or in two or three days. Such cases are apt to arise early in an epidemic. The patient has a violent chill; delirium occurs early; the headache is less intense, or at any rate gives way rapidly to stupor and coma. The pulse is frequent and feeble; there may be no rise of temperature, the skin being cool, clammy, and cyanotic. Local or general convulsions may occur. The eruption may be purpuric, and even ecchymoses may occur. A *mild* form usually occurs late in epidemics, it closely resembles the nervous type of influenza, and could easily escape recognition except during an epidemic.

An *abortive* form, ending in recovery in two or three days, and an *intermittent* form, with exacerbations on alternate days, have been described.

The *duration* of the disease is from a few hours to two or three months. In ordinary favorable cases there is decided improvement toward the end of the first week, and convalescence is established in two weeks. It may become chronic and last for weeks, or, as already stated, may be fatal in a few hours. Relapses are common in some epidemics.

Complications.—The most frequent *complications* are those involving the lungs and heart, particularly pneumonia and endocarditis or pericarditis. Pneumonia often occurs so early that it is difficult to decide whether it is primary, with marked nervous symptoms, or is only a complication of the cerebrospinal fever. Nephritis also occurs.

The most frequent *sequels* are deafness, blindness, headache, and local palsies.

Laboratory Diagnosis.—The blood shows a pronounced hyperleukocytosis of the neutrophilic type. By blood-cultural methods or even in blood-smears the meningococcus can be demonstrated in a fairly large percentage of cases.

An absolute diagnosis of the condition may be made by lumbar puncture. The liberation of turbid fluid, escaping through the cannula under considerable pressure, is most suggestive, but still more so is the large percentage of polynuclear neutrophiles in the differential count of the stained specimen. If the flattened diplococci are found in the leukocytes the diagnosis is verified.

Diagnosis.—The diagnosis in the presence of an epidemic is not difficult, although an absolute diagnosis can only be made by lumbar puncture.

The epidemic form of meningitis must be distinguished from pneumococcus meningitis, tuberculous meningitis, and streptococcus meningitis, or more rarely scarlet fever, acute articular rheumatism, typhoid fever, and typhus fever which may at the onset suggest epidemic meningitis. If there is any doubt of the diagnosis, lumbar puncture should be immediately performed, as a good prognosis depends upon

an early diagnosis and application of proper therapeutics, in the form of Flexner's serum.

Acute Poliomyelitis.—An acute epidemic and endemic infectious disease, characterized by diffuse lesions anywhere in the nervous system, more especially by lesions in the anterior horns of the gray matter of the cord.

The disease is caused by an organism recently isolated by Flexner and Noguchi. The organism is transmitted by flies or by direct contact and enters the central nervous system by the way of the nasal mucous membrane. The disease is endemic, frequently becoming epidemic in the summer and autumn. It usually attacks children under six years of age.

Symptoms.—In the ordinary form, after an incubation period of five to ten days, the child is seized with chills, fever, headache, and with rapidly developing paralysis, which attains its greatest height in a very short time. The paralysis is extremely irregular and various muscle groups may be affected. The muscles of the legs are more frequently involved than the arms, but the extremities may be affected without any regularity as an arm on one side, a leg on the other side. The muscles are usually affected in functionally similar groups, such, for example, as the flexors of the upper arm. Pain in the muscles is usually absent, though at times may be so pronounced as to simulate a polyneuritis. The affected muscles are flaccid, atrophy rapidly, and during the second week begin to show the reactions of degeneration. When atrophy is complete all electrical response is lost, although many of the muscles recover. Secondary contractures and deformities (club-feet, scoliosis, etc.) gradually appear if the atrophy is complete.

ATYPICAL TYPES.—The above type is the usual manifestation of the disease. Other types that may occur, particularly during an epidemic, are:

1. *Abortive Type.*—The child is seized with fever and symptoms of cerebrospinal irritation without paralysis. The attack passes off in a few days, and unless an epidemic is present the diagnosis is not made.

2. *Progressive Ascending Type* (simulating Landry's paralysis).—The paralysis begins in the lower extremities, rapidly ascends and the child dies about the fifth day with symptoms of bulbar paralysis.

3. *Meningeal Type.*—The symptoms are those of cerebrospinal fever, from which condition acute poliomyelitis is only differentiated by the results of lumbar puncture.

4. *Bulbar Type.*—The symptoms are those of paralysis of the bulbar nerves plus the acute symptoms of the infection.

5. *Cerebral Type.*—In a certain number of cases following the onset of the disorder, the paralyzes are those of a cerebral hemiplegia.

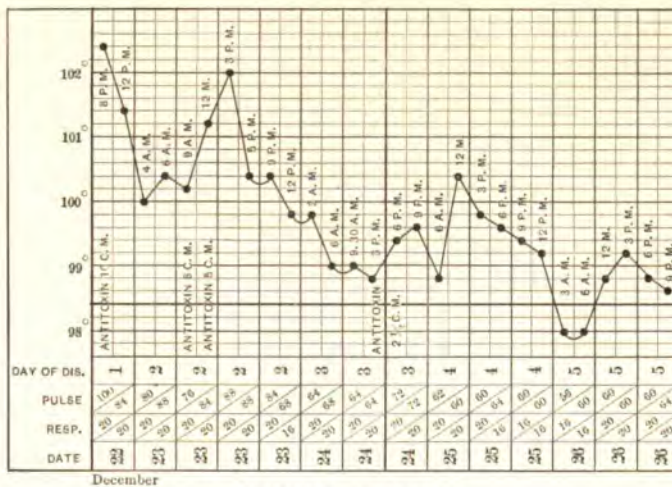
Diagnosis.—The diagnosis of the ordinary type is usually made out difficulty. The irregular types are diagnosticated with care. The diagnosis of these types is usually based upon the course in the course of epidemics, and upon the fact that the globulin reaction of Noguchi (*q. v.*).

Diphtheria.—An acute, infectious, and contagious disease, sporadic and epidemic, caused by the Klebs-Loeffler bacillus, occurring especially in children from one to six years of age, and characterized by insidious or abrupt onset, with moderate fever, and the development upon the fauces or upon any abraded surface of a grayish-white false membrane, which has a tendency to extend, especially to the larynx.

Symptoms.—The period of *incubation* varies from a few days to two weeks, or perhaps longer in exceptional cases. As a rule it is less than a week.

The *onset* in mild cases is deceptively free from positive symptoms. The child is languid, perhaps slightly chilly, and has a little fever, with thirst, impaired appetite, and discomfort in swallowing. Unless the nature of the trouble is suspected, the child is not thought ill

FIG. 173



Diphtheria. (Original.)

enough to be kept indoors. The throat is slightly inflamed, especially about the tonsils. The child may protest that there is no pain on swallowing. In from twelve to twenty-four hours after the onset, sometimes later, a grayish pellicle will be found on the tonsils, and the cervical glands will be swollen. In more severe cases the disease begins with chills or chilliness, followed by a rise in temperature to 102° or 104° F., sore throat, and sometimes vomiting. Convulsions and delirium may occur. Headache, thirst, disgust for food and aching in the back and limbs may be complained of. Prostration is often very pronounced from the beginning.

The *temperature* usually falls on the second or third day, but this does not indicate either a favorable or unfavorable end. A temperature *slightly* above normal is not uncommon in profound toxemia. The

unassociated with a septicemia, just as it is impossible to have a septicemia without a concurrent toxemia. Likewise, the term *sapremia*, the absorption of the products of putrefaction, is inadvisable, as such a process is always associated with the implantation and growth of bacteria in the tissues unless, of course, the products of decomposition are ingested in the food and absorbed from the alimentary tract, in which case infection does not occur.

Toxemia.—*Causes.*—A toxemia arises as a result of the invasion of any pathogenic bacteria. The severity of the symptoms, however, vary according to the amount and character of the toxin elaborated. Thus in diphtheria, tetanus, erysipelas, and pneumonia, severe constitutional symptoms arise, as a result of the powerful action of the absorbed toxins elaborated by these germs. Conversely in gonorrhea constitutional symptoms are absent as the action of the gonotoxin is limited to the urethra. Other causes of toxemia are the absorption of localized products of putrefaction in which the toxins elaborated by the bacteria play a minor part in causing toxemia, *e. g.*, retained placenta, gangrene, localized suppurations, and necrosis.

Symptoms.—Chilliness or chills, fever, malaise, prostration, nervous manifestation, and rapid pulse are the clinical evidences of toxemia. The rapidity and weakness of the pulse is the best indications of the severity of the toxemia. Some toxins exert certain definite effects upon certain parts, *e. g.*, tetanus and diphtheria upon the motor neurones and nerves, respectively.

Septicemia.—*Causes.*—Any infection in which the bacteria enter the blood current may be the cause of septicemia but commonly the term is applied to a streptococcic or staphylococcic infection. At times the cause of the infection, an injury, puerperal sepsis, abscess, or an acute infectious disease may be quickly discovered. Again a so-called cryptogenetic septicemia, in which there is no discoverable source of infection, may be present. This is frequently a terminal infection of an acute or chronic disease, although it may occur in those who are apparently healthy. In severe forms of the specific infectious diseases there is often a mixed infection, streptococci or staphylococci occurring in the blood the same time as the specific organism.

Symptoms.—Excluding the specific infectious diseases, which are individually discussed elsewhere, the symptoms of a pyogenic septicemia are those of a severe toxemia plus the local symptoms arising at the focus of infection (when discoverable), but which are frequently entirely obscured by the systemic invasion. The onset is marked by a chill which is followed by a pronounced rise of temperature. The temperature is extremely irregular as a rule, the rises often being accompanied by chills and followed by sweats. There is more or less prostration and marked constitutional reaction. Malaise, anorexia, nausea, constipation, and nervous symptoms are common. There is considerable anemia and consequent pallor of the skin and mucous membranes. The pulse is rapid, over 120, and weak; the tongue dry

and coated. Objective symptoms otherwise are usually absent, although purpuric spots are at times seen in the skin. A mixed infection, occurring in the course of the specific infectious diseases, notably typhoid fever and tuberculosis, will often markedly obscure their clinical picture. The course of the disorder is usually prolonged, although death may occur in a few days. Recovery is relatively frequent, much more so than was one time thought possible, especially in the streptococcic septicemias from local infections.

Pyemia.—*Causes.*—Pyemia is septicemia complicated by multiple abscesses; therefore any condition capable of causing septicemia may cause pyemia. The important factor in the causation of pyemia is the presence of infected emboli circulating in the blood. They may be simply masses of bacteria, but much more frequently the emboli arise as a result of inflammation of the veins (phlebitis, rarely the arteries) in the suppurative focus which in turn causes formation of thrombi, from which small infected particles are carried into the general circulation as infected emboli, which become impacted in the small capillaries of distant parts to form metastatic or embolic abscesses. The location of these abscesses depends largely upon the original focus of infection; thus if in the portal system, multiple abscesses of the liver will develop, associated at times with suppurative pylephlebitis; if in the general venous system, the emboli will lodge in the lungs; and if in the heart or arteries, emboli enter the general arterial circulation causing renal, splenic, arthritic, muscular, and cerebral abscesses. Bacteria circulating in the blood may lodge in areas of lessened resistance without the intermediation of emboli. Thus they frequently lodge in a heart valve, the seat of chronic disease, here causing the productions of infected vegetations which are washed off into the circulation to act as emboli.

Symptoms.—The constitutional symptoms are those of an exaggerated and profound septicemia. Exceedingly irregular fever is present. The rises in temperature are accompanied by a rigor and the falls by profuse sweats. The prostration is extreme, and the patient soon passes into the typhoid state, and dies of exhaustion in two to three weeks, if some intervening complication does not cause death sooner. In the so-called chronic pyemia there are fewer and usually superficial abscesses, and the disease runs a longer course.

The local symptoms and signs depend upon the location of the metastatic abscesses; if in the lungs, cough, dyspnea, pleuritic pain, and purulent expectoration; in the liver or spleen, enlargement and tenderness of the organ; in the kidney, purulent urine and lumbar pain and tenderness; in the pericardium, pain; in the endocardium, the signs of ulcerative endocarditis; in the joints, pain, tenderness, and swelling; in the eye, sudden loss of vision; in the superficial structures, the usual signs of abscess, pain, tenderness, redness, swelling, and fluctuation.

Laboratory Diagnosis of Septicemia and Pyemia.—Excepting the common manifestations of septicemia, *i. e.*, most of the specific infectious diseases, which are rarely accompanied by pyemia, the organisms found by blood culture are usually streptococci or staphylococci.

The blood shows a severe secondary anemia, a hyperleukocytosis, and the presence of Simons' septic factor, an increase of neutrophilic and decrease or absence of eosinophilic leukocytes. Iodophilia is also present, and usually hemolysis can be demonstrated grossly by the reddish tinge of the blood serum.

A bacteriuria is frequently present, and pyuria if there are metastatic renal abscesses.

Diagnosis of Septicemia and Pyemia.—In suspected cases a focus of infection must always be carefully and thoroughly sought for. A small infection of the extremities may be overlooked or disregarded unless the red streaks of inflamed lymphatics are observed. Of the more common sources of infection, these may be mentioned, septic postpartum endometritis; suppurating wounds, cellulitis; suppurative mastoiditis either with sinus thrombosis or with meningitis; osteomyelitis, periostitis, and septic arthritis; gonorrheal infections and chancroids; tonsillitis, sinusitis; and ulceration of the intestines, appendicitis, and suppurating hemorrhoids. Septicemia or pyemia may simulate acute miliary tuberculosis, malaria, typhoid fever, and influenza, and in turn they are frequently simulated by an abscess of the genito-urinary or alimentary tract, an empyema, Charcot's intermittent fever, or rapidly developing cachectic conditions.

The differentiation between severe toxemia and septicemia is only made by blood culture. Pyemia is usually not differentiated from septicemia until the occurrence of the metastatic abscesses.

Glanders.—An infectious disease, transmitted from horse to man, caused by the *Bacillus mallei*, appearing in an acute and chronic form, and characterized by the appearance of nodules in the nose (glanders) or beneath the skin (farcy), terminating in death in from one to four weeks in the acute form, or in 50 per cent. of the chronic cases in three or four months.

Symptoms.—In *acute glanders* the onset is marked by headache, slight fever, and pain in the limbs. In a day or two the nodules appear in the nose, the mucous membrane becomes swollen, the external surfaces reddened, and the cervical glands enlarged. These nodules rapidly break down and a mucopurulent discharge becomes evident, soon becoming purulent and fetid. Sometimes a diffuse redness, resembling erysipelas, spreads from the affected point. At the same time there appears an eruption, consisting of papules which rapidly become pustules which rupture and give vent to a thin purulent discharge. The patient gradually sinks into a septicemic condition, with irregular fever, dry, brown tongue, albuminuria, delirium, coma, and collapse.

In *acute farcy* there are similar constitutional symptoms, but the primary localization takes place beneath the skin. Pronounced ulcera-

tion rapidly develops, with here and there nodular enlargements (farcy buds) in the reddened and swollen surrounding lymphatics, which go on to suppuration. Abscesses may appear in the muscles and the joints become painful and tender. Septicemic symptoms rapidly develop.

In *chronic farcy* there are deep ulcers upon the hand, face, forehead, or elsewhere. In still another form (pneumonic) the prominent symptoms are cough, bloody expectoration, hoarseness, fever, and emaciation.

Laboratory Diagnosis.—The *Bacillus mallei* can frequently be demonstrated by cultural methods in the nasal discharge and in the pus from the ulcerations. Injection of this matter or of the culture from it into a guinea-pig peritoneally will cause, in thirty-six to forty-eight hours, a well-marked periorchitis. The diagnostic sign is the tumefaction of the testicles. Hypodermic injections of mallein analogous to tuberculin, will produce a reaction similar to that of tuberculin in the tuberculous.

Diagnosis.—Acute glanders is to be distinguished from rheumatism, septicemia, pyemia, variola, and erysipelas. The chronic forms may suggest the ulcerations of tuberculosis or syphilis or in those cases with pronounced respiratory symptoms, pulmonary tuberculosis may be mimicked.

Cholera.—An acute, infectious disease, caused by the comma bacillus of Koch, and characterized by vomiting, copious discharge from the intestines, an early collapse, and painful muscular cramps.

Symptoms.—The period of *incubation* is probably short in the majority of cases, lasting only a few days. There are usually no definite symptoms during this time.

Preliminary Stage.—The first stage, that of premonitory diarrhea, is better regarded as the beginning of true cholera. It is characterized by profuse watery stools of a yellow or light yellow color and alkaline reaction. From six to a dozen of these passages occur in twenty-four hours. The patient feels faint and exhausted after them, and may suffer from nausea, but vomiting is not usual. The temperature is normal or slightly depressed. This stage may last from two days to a week. In some cases it is absent, and the second stage sets in abruptly.

Collapse Stage.—The patient is seized with vomiting, which is at first bilious, but rapidly loses all color and becomes like rice-water. The stools likewise resemble water in which rice has been soaked. This fluid seems to well up and regurgitate rather than to be vomited from the stomach, and to gush in quantities of a quart or two from the anus. Sometimes vomiting and diarrhea occur at once. The patient has unquenchable thirst, and is tortured with painful cramps of the toes, legs, belly, and diaphragm. As the discharges continue the patient becomes more and more exhausted; the nose is pinched and twisted, the eyes sunken, the lips bluish, and the whole body may shrink beyond recognizable proportions. The skin is cold and moist, the breath icy, and the temperature under the tongue is sometimes as

low as 78° F. In the vagina and rectum it may be normal or slightly above normal. The patient, however, often has a sensation of heat. The pulse is very small and feeble, its rate 100 to 120. In some cases the patient dies before purging occurs (cholera sicca). Mild cases (cholerae) with but slight collapse are usually seen at the end of epidemics.

Reaction Stage.—From this collapse and algid condition the patient may slowly emerge, the skin becoming less cold, the cramps less severe, with a return of the secretion of the urine, and a cessation of vomiting and decrease of diarrhea. The reaction, however, may simply introduce a low typhoid condition, with fever, dry, brown tongue, subsultus, low muttering delirium, and coma.

Complications and Sequelæ.—The most frequent are ulcerations, bed-sores, and parotitis; uremia, diphtheritic inflammations of the mucous membranes, pleurisy, and pneumonia occur rather commonly. During convalescence, painful tetanic spasms of the legs and arms may be seen.

Laboratory Diagnosis.—A relative polycythemia develops as a result of the excretions of such large quantities of the body fluids. The leukocytes are not only relatively but actually increased. An agglutinative reaction appears early.

In the feces, the comma bacillus may be demonstrated, as a rule, by appropriate methods in a few minutes, always by cultural methods in twenty-four to forty-eight hours. The secretion of urine is much diminished or completely suppressed. An albuminuria is always present, and if a complicating nephritis develops, red-blood cells and numerous casts will be found.

Diagnosis.—In doubtful sporadic cases the diagnosis may frequently only be made by bacteriological studies. During an epidemic there is usually but little question as to the diagnosis.

Cholera nostras or very rarely *arsenical* or *other toxic gastro-enteritides* may so simulate cholera that the diagnosis can only be made by bacteriological methods.

Bacillary Dysentery.—An infectious disease, caused by the *Bacillus dysenteriae*, and characterized by inflammation of the intestinal tract, chiefly the colon, attended by the symptoms of intestinal catarrh in intense degree, with mucus and bloody discharges and the general symptoms of fever and prostration, followed by extreme exhaustion.

Symptoms.—After an incubation period of two or three days the onset is marked by the occurrence of colicky pain, at first localized around the umbilicus; and later most marked in the course of the colon. The bowel movements are frequent, preceded by constant desire and attended by extreme tenesmus. The stools, which are first fecal and fluid, soon become scanty, and consist almost entirely of mucus and blood. The symptoms of local proctitis are severe; there is a sensation of a hot mass in the rectum. There may be strangury, and prolapse of the rectum may ensue.

The acute pain and frequent evacuations continue, the skin becomes hot and dry; the tongue is red and raw; thirst, nausea, and occasionally vomiting occur. The temperature continues at about 103° F.; the pulse is rapid. The patient is weak and restless. If the disease is severe from the start, or the course unfavorable the stools may contain pure blood, or they may be dark in color, and contain shreds of membrane, discharged involuntary. The tossing and restlessness are characteristic, and are attended by sighing and some dyspnea. The course of the disease is from twenty-eight to forty-two days. A fatal result occurs in about 25 per cent. of the cases.

Chronic Dysentery.—Convalescence is slow in most cases, and is usually associated with extreme anemia. In a few cases the severe symptoms never appear or disappear, but moderate diarrhea continues for a long time, the feces being mixed with mucus and pus from the ulcerations. The patient is anemic, weak, and emaciated, eventually dying from inanition or possibly as a result of intestinal structure.

Other Clinical Types.—Acute catarrhal dysentery in the great majority of cases is probably due to the *Bacillus dysenteriae*. Diphtheritic dysentery is a type of bacillary dysentery characterized by formations of a pseudomembrane, attached to the mucosa of the large bowel.

Complications and Sequelæ.—Of these, parotitis, peritonitis, pyelophlebitis, nephritis, pericarditis, painful and swollen joints and abscess formation are the most common.

Laboratory Diagnosis.—The blood picture is that of a severe secondary anemia associated with a polymorphonuclear leukocytosis. An agglutination reaction with the *Bacillus dysenteriae* is possible by the end of the second week. In the first week of the disease the organism may be isolated from the feces by cultural methods.

Diagnosis.—The diagnosis of dysentery, *per se*, is readily made. To positively differentiate the several forms of dysentery, however, is impossible unless careful laboratory studies are made.

The Plague.—An acute, infectious, and contagious disease, caused by the *Bacillus pestis* of Kitasato, occurring in epidemics, characterized by high fever, sometimes by petechia and other hemorrhages, and, in cases which last long enough, by buboes. The death-rate is very high.

Symptoms.—The period of incubation is from two to seven days. The invasion is marked by lassitude, languor, headache, and dizziness. The stupid aspect and staggering gait may lead to the belief that the patient is drunk. Chill or chilliness soon supervenes, followed by fever, which often rises to hyperpyrexia, and is accompanied by unquenchable thirst, and sometimes nausea and vomiting. Delirium and a typhoid condition follow, with a marked tendency to failure of the circulation and collapse. If the patient survives until the second or third day, glandular swellings develop in the groin or axilla, or at the angle of the jaw. Often they have to be sought for to be found. Sometimes they are prominent, and are followed by suppuration and even ulceration. Carbuncles are much rarer manifestations than buboes.

Petechiæ, vibices, hemorrhages into the kidney, and bloody vomit occur in the worst cases.

The clinical divisions are (1) *pestis minor*, in which slight fever, some glandular swelling, and possibly suppuration occur; (2) *pestis major* (described above), in which the development of buboes is the chief characteristic; (3) *septicemic plague*, in which the attack is fulminating, death resulting in from eighteen to sixty hours; (4) *pneumonic plague*, in which the predominating symptoms are those of a grave bronchopneumonia; (5) *intestinal plague*, in which abdominal and enteritic symptoms are pronounced.

Laboratory Diagnosis.—A hyperleukocytosis is present. The causative organism may be found by blood culture and in the buboes, by puncture, or in the urine, feces, and sputum.

Diagnosis.—In all cases cultural methods should be employed and guinea-pigs inoculated.

Leprosy.—A chronic, infectious disease, caused by the *Bacillus lepræ*, characterized by the development of tubercles, or anesthetic patches and neuritis.

Symptoms.—The disease develops slowly and insidiously. Sometimes the first skin lesion is a crop of bullæ. More commonly there appear reddish or violet-colored patches, varying in size from one-quarter inch to two or three inches in diameter, and becoming of a darker hue later. The next step is the formation of nodules, which are characteristic of the disease. These may develop upon the patches already described, or in other places. They vary in size from that of a pea to that of a bird's egg or larger. They are most common upon the face and extensor surfaces of the arms, legs, fingers, and toes. The tubercles consist of an infiltration into the true skin; they are raised, firm, relatively painless, and vary in color from red to copper. The face is sometimes characteristically distorted into a fierce expression (leontiasis). The tubercles may become absorbed and leave atrophic areas, but generally they break down into eroding ulcers, which slowly burrow and increase in extent, eating off a portion of the nose, fingers, hands, and feet, and exposing muscles, tendons, nerves, bloodvessels, and bones. Tubercles form also upon nerve trunks, and ulcers upon the mucous membranes. (See the Nose and Larynx.) In other cases, or in combination with the tubercles, especially upon the trunk and limbs, there are *anesthetic* areas. Ulcers may follow without the previous occurrence of tubercles, giving rise to contractures and necrosis, and resulting in marked deformities of the extremities. With the anesthetic patches are associated crops of *bullæ*, and *neuritis*.

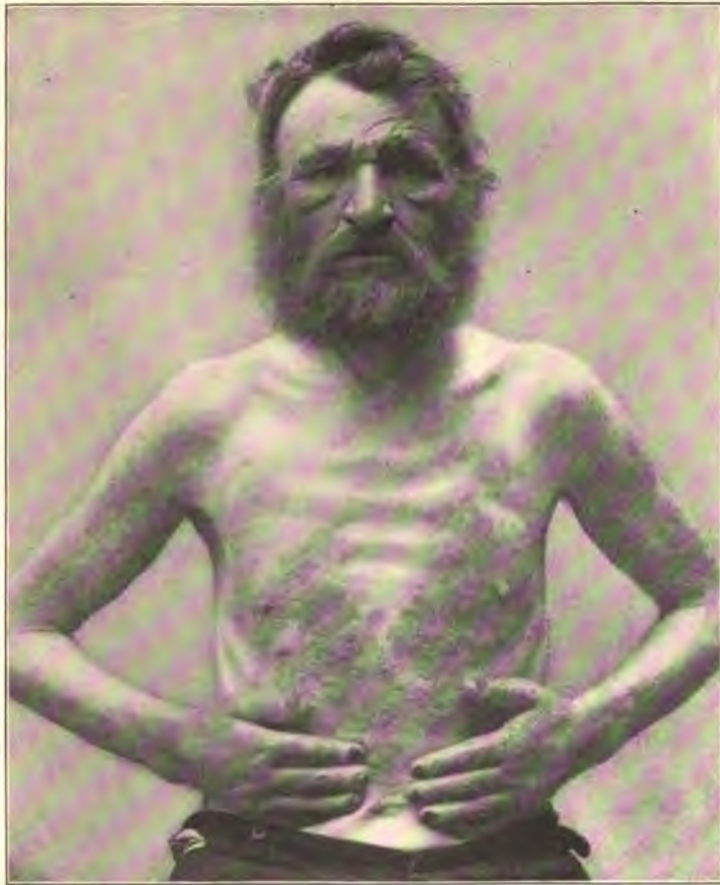
The further peculiarities of the disease are its long duration; its slow progress interrupted by apparent healing of some of the ulcers; its afebrile course (the temperature is slightly subnormal); its comparative painlessness; and the slight impairment of the general health.

Death results from gradual wasting, or is hastened by some intercurrent affection.

Laboratory Diagnosis.—The blood is anemic in direct relation to the severity and stage of the local lesion.

The *Bacillus lepræ* is found in the pus of the ulcers and other lesions. It is extremely difficult to artificially cultivate the organism and impossible to cause the infection of guinea-pigs by injection.

FIG. 174



Leprosy.

Diagnosis.—The lesions are usually so typical that the diagnosis is simple. In rare cases lupus and tubercular syphilides are only excluded by laboratory studies.

Tetanus.—An acute infectious disease, caused by the *Bacillus tetani*, the essential characteristic of which is persistent tonic spasm of the

muscles of the jaws (lockjaw) and of the spinal and thoracic muscles, associated with general clonic convulsions.

Symptoms.—After an incubation period of one or two weeks, during which time prodromata are usually absent, the disease begins with stiffness of the jaws, which steadily increases, until within a few hours there is complete tonic spasm. The neck muscles, and then those of the spine and trunk, become rigid, so that the body is arched backward and may rest upon the heels and head (opisthotonos). The facial muscles share in the spasm, and by their contraction produce a horrid, grinning countenance (risus sardonicus). The contracted muscles become painful, and there is also epigastric pain. The rigidity is persistent, but is interrupted by exacerbations in which the phenomena already described are exaggerated, and, in addition, respiration is embarrassed, the face becomes livid, the skin bathed in sweat, and the patient is further distressed by increased pain in the affected muscles. The body may be bent forward (emprosthotonos) or laterally (pleurothotonos). The temperature is not constant. It may remain normal, be moderately elevated, or hyperpyrexia may be present, especially toward and after the end of fatal cases. The spasm ceases during sleep, but subsequently returns.

Laboratory Diagnosis.—A moderate leukocytosis is found. Injection of pus from the primary wound, if it contains the bacilli, causes characteristic convulsions in the guinea-pig.

Diagnosis.—Tetanus must be distinguished from *strychnine-poisoning*. In the latter the jaw muscles are never involved early, if at all, and the muscles are relaxed between the paroxysms. It is distinguished from tetany by the history and the distribution of the spasm, which in tetany is confined to the extremities and from *hydrophobia* by the spasms coming on during attempt at swallowing.

Ephemeral Fever.—**Febricula.**—**Simple Continued Fever.**—A non-contagious fever lasting from one to twelve days, not dependent upon any known specific cause, and not attended by definite local lesions. The chief feature is the continued elevation of temperature. If persistent for only twenty-four to forty-eight hours it is spoken of as *ephemeral fever*; a fever for seventy-two to one hundred and forty hours is called *febricula*; longer than this as simple continued fever.

The onset of the disease is abrupt. There may be a chill, or in nervous children a convulsion; but these are rare. The temperature rises rapidly from 102° to 104° F., accompanied by headache, thirst, restlessness or drowsiness, loss of appetite, a coated tongue, constipation, and occasionally nausea. The urine is scanty, and sometimes there is a heavy deposit of urates. There may also be more or less muscular soreness.

The *diagnosis* from other fevers and febrile affections is made in the absence of any characteristic eruption, of enlargement of the spleen and liver, and of any lesion, such as endocarditis, bronchitis or pneumonia.

Actinomycosis.—An infectious disease of cattle, caused by the *Streptothrix actinomyces bovis*, occurring occasionally in man, attacking especially the lower jaw, lungs, and intestines, and characterized by a long duration, by the development of metastatic nodules, and by pyemic symptoms.

Symptoms.—The primary seat of invasion in the majority of cases is in the buccal or pharyngeal cavities where a slowly growing, slightly painful tumor develops. Bones are affected as well as soft tissues. These become swollen and suppurate, leaving persistent sinuses and fistulæ; the fungus is commonly found in the discharge.

From here the organisms may be carried to the respiratory and gastro-intestinal tracts or the disease may be primary in these systems.

FIG. 175



Case of actinomycosis.

RESPIRATORY FORM.—Actinomycosis of the lung may be divided into three stages; a latent stage, when the lung proper is affected; an active stage, when the extension to the pleuræ and chest wall takes place; and a final or chronic stage, when perforation and the formation of a thoracic fistula occurs and the adjoining organs become affected.

The symptoms of the *first stage* are those of chronic bronchial catarrh.

The symptoms of the *second stage* are those of pleurisy, with adhesions, with or without effusions, plus a superficial swelling on the chest wall. Fever and pain accompany these processes. The course of the disease at this time may extend over many months in contradistinction to empyema on the one hand and carcinoma on the other.

In the *final stage*, ulceration of the swelling is seen in many places, a fistula forms, and the disease extends to adjacent structures.

GASTRO-INTESTINAL FORM.—The symptoms of this form are those of a persistent and aggravated gastro-enteritis plus a local tumor or swelling of the abdominal wall, commonly in the ileocecal region, which subsequently ruptures. Symptoms of appendiceal abscess, hepatic abscess, or perforative peritonitis may develop or the disease may run a chronic course with anemia, cachexia, septic symptoms, and symptoms of amyloid degeneration.

Metastasis.—Secondary infection may occur and symptoms of pyemia develop. Metastasis to any organ may occur, with resulting local symptoms. The duration depends upon the organs involved in metastases.

Laboratory Diagnosis.—A pronounced secondary anemia of the chlorotic type develops usually with an accompanying leukocytosis. The pus, and at times the sputum, is most characteristic. Nodules of gray or yellow color, sulphur granules, the size of a poppy-seed, can be seen with the naked eye. With a low-power these particles appear as aggregations of clubbed threads, which with a higher power are seen to be arranged in masses radiating from a common centre. Each separate thread has a clubbed end. They have high refractive power. The centre of the masses is occupied by a net-work of fibers. If the masses be broken up, numerous club-shaped forms are seen at the periphery. The urine in the latter stages of the disease shows the signs of amyloid degeneration of the kidney. Sudden appearance of pus, much albumin, and many casts usually means metastatic involvement of the urinary tract.

Diagnosis.—Actinomycosis so frequently simulates other chronic inflammatory conditions and tumors that a positive diagnosis is only made by the discovery of the organism in the pus.

Streptothrichal Infections.—The pathogenicity of streptothrices and their relation to infections and lesions described are very evident, though cultural and animal experiments are lacking in some of the earlier observations. It is clear that actinomycosis and Madura foot can no longer occupy their isolated positions as examples of streptothrichal infections. The predilection of these organisms for certain regions or systems of the body is very apparent, the *lungs*, the *brain*, and the *skin* being most frequent affected; the brain, however, is more often invaded by metastasis in cases of primary pulmonary or bronchial gland infections.

The *pulmonary lesions* in which streptothrices have been found either alone or with other organisms show considerable variety: broncho-pneumonia, extensive solidification, abscess, bronchiectasis, empyema, and necrotic bronchitis.

The lesions in the *nervous system*, practically always metastatic and secondary to pulmonary or bronchial gland disease, have been abscess, meningitis, or large areas of softening from thrombosis. An *abscess* of the *kidney* was noted in one instance. Naunyn found in his case

an *endocardial excrescence* containing a streptothrix. From a clinical standpoint but little that is distinctly new is presented in streptothrical infections. The cutaneous lesions of Madura foot, the cutaneous expression of actinomycosis, the actinomycotic-like skin infection of Fullerton, the erysipeloid of Rosenbach show distinct differences. The pulmonary infections with their secondary accidents are all more or less hidden under the mask of a tuberculous-like process, sometimes termed *pseudotuberculosis* or resemble some one or other of the number of pulmonary lesions, such as *abscess*, *gangrene*, or *bronchiectasis*, but the detection of the streptothrix and its mycelial masses is as diagnostic as the demonstrations of tubercle bacilli.

CHAPTER XXXII

DISEASES DUE TO ANIMAL PARASITES

I. DISEASES DUE TO PROTOZOA

Relapsing Fever.—An acute infectious and contagious disease, usually epidemic, caused by the spirochete of Obermeier (European form), transmitted probably by lice and bed-bugs, and characterized by the sudden onset of a febrile period lasting five or seven days, which is followed by an intermission lasting usually a week, and this in turn by a relapse lasting three days. Relapsing fever occurring in other countries, presents much the same clinical symptoms as the European form, although the spirochetes causing the other forms, differ morphologically from the *S. obermeierei*.

Symptoms.—The stage of *incubation* lasts from five to eight days. The *invasion* is sudden, with a chill or chills, frontal headache, pain in the back and limbs, vertigo, and great physical weakness. The temperature rises very rapidly, reaching 105° to 106° F., and the pulse is correspondingly accelerated. Epistaxis sometimes occurs. The appetite is usually lost, thirst is intense, and the bowels constipated. A mild catarrhal jaundice is not infrequent. Nausea and vomiting are prominent symptoms in children. Tenderness with pain in the epigastrium is frequent and a moderately enlarged spleen may be palpated. On the fifth or seventh day a decided *crisis* occurs, which, however, is sometimes deferred until the tenth day. The *temperature* within twelve hours falls from 106° or 108° F. to or below normal, and the subjective symptoms rapidly disappear. The crisis is marked most frequently by a profuse sweat, sometimes by diarrhea, epistaxis, metrorrhagia, or intestinal hemorrhage. The patient now enters upon rapid convalescence without fever. On the seventh day from the crisis, however, a sudden relapse occurs, with a repetition of the symptoms of the first attack. The temperature may be higher and the febrile symptoms more severe, but the duration is shorter—only three or four days. The spirochetes which apparently disappeared in the apyretic interval are again found in abundance. A second crisis, with its associated symptoms, now occurs. The spirochete again disappears, and in the majority of the cases there is no further bar to complete recovery. A second, third, and even a seventh relapse may occur. Organic lesions do not accompany or follow this disease, unless they have occurred as complications; but even in ordinary cases the patient is left weak, anemic, and in poor condition.

Relapsing fever occurs at all ages, but is most common in adults. The duration varies according to the number of paroxysms. If there is only one, it is about eighteen days. About 4 per cent. of the cases terminate in death. Under the name "bilious typhoid" a malignant form of relapsing fever has been described. It is characterized by intensity of the symptoms of the ordinary form, the patient passing into the typhoid state and slowly dying.

Complications.—The most frequent complication is lobar pneumonia. Nephritis, cardiac weakness resulting in thrombosis and embolism, suppurative parotitis, rupture of the spleen, profuse epistaxis, abortion in pregnant women, and neuritis, although uncommon, deserve mention.

Laboratory Diagnosis.—The blood changes are those of a secondary anemia with moderate neutrophilic leukocytosis. The spirochetes can be demonstrated in blood smears, usually most readily during the febrile periods. The agglutination reaction can be employed at times successfully.

Diagnosis.—The diagnosis is based upon the occurrence of an epidemic, the characteristic and typical clinical course, and the examination of the blood. Relapsing fever is most likely to be mistaken for *typhus fever*, which occurs under similar conditions, but the blood examination will disclose the true nature of the disease.

Syphilis.—Syphilis is a chronic infectious disease which may be acquired or congenital, and is caused by the *Spirochaeta pallida*.

Acquired Syphilis.—This is characterized (1) by the initial lesion, or chancre, which appears usually in about three weeks after contagion, and in which the spirochete can be demonstrated by dark field illumination of fresh smears or in stained smears from the discharge; (2) by a period of latency generally lasting six weeks, but varying from one to three months; (3) by so-called secondary symptoms; (4) after an interval varying several months to twenty years, by so-called tertiary phenomena, which manifest themselves in some cases and are absent in others.

Course.—The course of syphilis in different attacks varies widely from a mild infection in which the early symptoms are almost unnoticed and the condition apparently remains latent until later years, when the nervous system may be attacked, to a malignant infection in which terrifically severe tertiary lesions follow immediately after or take place in the secondary manifestations of the disease.

The initial lesion (the chancre), appearing within a month after inoculation, starts as a small papule which goes on to ulceration and the surrounding tissue becomes hard and indurated. The glands of the lymphatics draining the region of the chancre become enlarged and hard. There are no constitutional symptoms as a rule.

Secondary Symptoms.—In an ordinary case of acquired syphilis, in about six weeks after the appearance of the chancre, the patient complains of languor, weariness, slight fever, pains in the bones, and impaired digestion, and shows a tendency to anemia. An *eruption* now

appears, coming out gradually during two or three weeks and persisting for about two months. It is most marked on the trunk and upper extremities, especially the chest and forehead (*corona Veneris*). The eruption may be roseolar, squamous, vesicopapular, papular, pustular, bullous, or tubercular. The color has been aptly compared to that of raw ham. The enlargement of the inguinal, epitrochlear, and post-cervical glands, which precedes the eruption, persists. Shallow *ulcers* with a sharply defined, grayish outline appear on both tonsils. They are painless and do not spread. Flat, grayish, raised, and macerated macules and papules with sharply defined margins may appear upon the pharynx, buccal surfaces, tongue, angles of the mouth, penis, vulva, vagina, and around the anus (mucous patches). Sometimes the hair becomes very thin and falls out, leaving the patient without eyebrows and more or less bald. *Iritis* and *retinitis* are usually later symptoms. Other symptoms occasionally occurring at this stage are *periostitis*, usually slight, and *onychias*. The most common of the symptoms enumerated are the eruption and the tonsillar ulceration.

The *tertiary lesions* of syphilis are the late *syphilides* (see Skin) and *gummata* of the skin, subcutaneous connecting tissue, muscles, or internal organs. In the brain and spinal cord gummatus tumors, gummatus meningitis, gummatus arteritis, and localized sclerosis are found. In syphilis of the lung we may find *gummata* scattered through the lung or a fibroid interstitial pneumonia beginning at the root of the lung. Diffuse syphilitic hepatitis or *gummata* may be found when the liver is affected. The rectum is the most common seat of syphilis of the digestive tract. Myocarditis and localized *gummata* and endarteritis occur in cardiac syphilis, while in vascular syphilis obliterating endarteritis and periarteritis are found. Syphilitic orchitis often occurs. Syphilis of a particular viscus is discussed more fully in the chapter devoted to disease of that organ. The so-called para- or metasyphilitic disorders appear for ten or fifteen years, or longer, after the initial sore and the subsidence of acute symptoms; the chief manifestations are tabes and paresis; long thought to be syphilitic in nature, they have proved to be so since the introduction of the Wassermann reaction.

Hereditary Syphilis.—At birth the syphilitic infant usually exhibits no evidence of its inherited taint. In the course of from one to twelve weeks it develops "snuffling" in breathing. An *eruption* soon appears, symmetrical in distribution. It is most frequently erythematous or papular, but it may be squamous, vesicular, pustular, or bullous. In hereditary syphilis the eruption is more apt to be moist and to affect the genitalia and flexures of the thighs than in acquired syphilis; it has, however, the same ham color as is seen in the acquired form. Coincident with the snuffles and the eruption appear *stomatitis* and *ulcers* at the angles of the mouth, and sometimes condylomata around the anus. Meantime the child has begun to waste, to be peevish, to be anemic, and gradually to assume the appearance of a wizened,

dried-up old man. As in acquired syphilis, *iritis*, although uncommon, and inflammation of the other structures of the eye may occur, but nodes and disease of the liver are rare.

The infant very frequently dies during this period from exhaustion and inanition.

If the child survives for a year the secondary symptoms usually disappear and the disease becomes latent, usually until the time of puberty, when the tertiary manifestation of the disease similar to those of acquired syphilis appear. In the meantime the patient may appear fairly well, but usually his development is retarded, and there is a tendency to anemia. Nasopharyngeal catarrh, flattening of the bridge of the nose, premature decay of the upper incisor teeth, and a protuberant forehead are frequent sigmata of syphilis. The teeth may be normal or may present the malformation of the two permanent upper central incisors known as "Hutchinson's teeth," which are peg-shaped, tapering from the gum downward with a single superficial vertical notch in the edge of the tooth.

Further, the patient may develop keratitis and other ocular changes, labyrinthine or cerebral deafness, syphilitic nodes and periostitis of the long bones, and ulcerations upon the legs.

Laboratory Diagnosis.—The blood shows the changes of a secondary anemia. Justus has shown that immediately after the first administration of mercury there is a rapid fall of hemoglobin.

The Wassermann reaction is a most valuable and trustworthy aid in the diagnosis of obscure syphilitic lesions. (See page 368.)

Recently Noguchi has devised a specific anaphylactic skin reaction, the luetin reaction, which does not require a trained laboratory technician to perform as does the Wassermann test. It consists essentially of the subdermal injection of sterile emulsion of *Spirochaeta pallida*, which in the presence of syphilis may cause one of the following reactions: (1) papular, in which a large raised indurated papule appears in from twenty-four to forty-eight hours; (2) pustular and similar to papular form for first three or four days, then multiple miliary vesicles appear which undergo central softening and become purulent in twenty-four hours; (3) torpid, no reaction for three or four days and then the appearance of small pustules. The reaction is positive in 100 per cent. of manifest tertiary syphilis and 94 per cent. of latent and 96 per cent. of hereditary syphilis. During the first and second stages the reaction is infrequent and when present, mild.

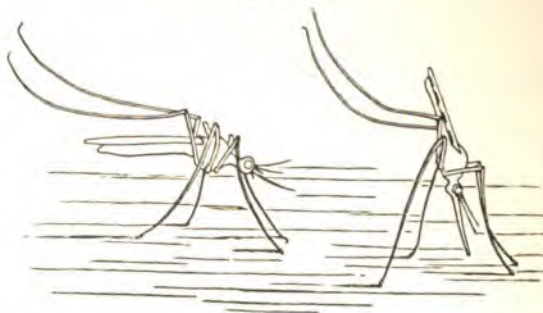
Diagnosis.—The diagnosis of *hereditary syphilis* is based upon the occurrence of snuffles and skin eruptions, and the existence of keratitis or of cicatrices, especially about the angles of the mouth. A history of repeated miscarriages is suggestive of maternal syphilis. The diagnosis of *acquired syphilis* is based upon the history of chancre, when that history is obtainable; upon the existence of polymorphous eruptions, or of non-traumatic ulcers upon the legs of young adults, or of scars in the groins or over the tibia, or of nodes, or of alopecia

associated with sore throat or mucous patches. The presence of obscure disease of the bones, glands, or spinal cord should lead to the search for a possible syphilitic infection.

The Malarial Fevers.—A group of infectious diseases due to the invasion of the blood by animal parasites belonging to the Sporozoa (suborder Hemosporidia, genus *Plasmodium*), and clinically characterized either by the production of periodical paroxysms of chills, intermittent fever, and sweats or by remittent or continuous fever, with irregular chills and sweats.

Malarial fevers, while most prevalent in tropical and subtropical regions, are found also throughout the temperate zone, especially in autumn and spring, and are most common in the United States in the Southern and Southwestern States. Conditions that especially favor their development are marshes and collections of stagnant water which acts as breeding places for the mosquito, the anopheles, which transmits the disease to man.

FIG. 176



Culex and anopheles. Anopheles is recognized by its spotted wings and tilted attitude.

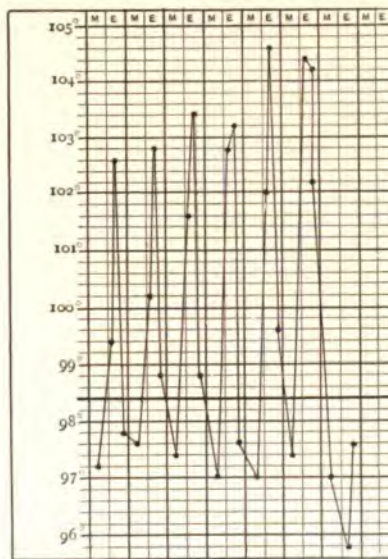
Intermittent Fever.—This is a type of malarial fever in which the temperature remains normal between the paroxysms.

The paroxysms, due to the segmentation of the parasites, occur periodically every other day (tertian type) if the infection is from a single group of *Plasmodia vivax*; every day (double tertian or quotidian type) if from two groups of the *Plasmodia vivax*. On the other hand if the *Plasmodia malarie* cause the disease, the paroxysm occurs every third day (quartan type), unless there is a double or triple group of *Plasmodia malarie*. While the rule is for the paroxysm to occur periodically at the same hour, if the disease is growing worse the second paroxysm may occur an hour or so earlier (anticipation), or, if it is growing better, an hour or two later (postponement). The double tertian infections are slightly more common than the single tertian, while the quartan variety is rare.

Symptoms.—A malarial paroxysm is characterized by (1) chill, (2) fever, and (3) sweating, occurring in the order named and in immediate succession.

The *incubation-period* probably varies widely, from a few days to months. The prodromal symptoms consist usually of malaise, headache, and constipation.

FIG. 177



Intermittent fever. Temperature every six hours. Morning and evening temperature; highest at chill.

The *onset* of a typical malarial paroxysm is marked by chilly sensations. Soon a decided chill sets in, the patient shaking violently. The face is pale and pinched, the lips blue, the nose pointed; as the chill becomes worse the teeth chatter, the whole body feels cold and shakes, the skin feeling rough, dry, cold, and harsh. The finger nails and toe nails are blue, the skin being wrinkled upon the palmar and plantar surfaces.

Nausea and vomiting are not uncommon. The spleen is perceptibly enlarged, and frequently also the liver.

Although the surface temperature is depressed, the internal *temperature* is rising, and may be two or three degrees above normal. By degrees the severity of the chill abates. As reaction sets in the surface bloodvessels dilate and the skin becomes flushed. The temperature continues to rise, often reaching 103° to 106°, pulse and respiration increasing correspondingly in frequency. The patient complains of a throbbing, dizzy headache, and vomiting may recur. The temperature

now begins to fall, and the sweating stage succeeds. The perspiration becomes more and more profuse, until the whole body is drenched with it. All the subjective symptoms vanish with wonderful rapidity, and the patient, with the exception of exhaustion, seems to be restored to complete health. The hot stage lasts from one to two hours, the cold stage from three to eight hours, and the sweating stage from two to six hours. During the entire paroxysm the mind remains clear.

In the interval between paroxysms the patient is free from fever, but is weak, and has impaired appetite and constipation.

The chief *objective symptom*, apart from the phenomena of chill, fever, and sweat, already described, is the occurrence of plasmodia in the blood and the enlarged spleen.

Remittent Malarial Fever—Estivo-autumnal Type.—A type of malarial fever due to the *Plasmodia falciparum* and characterized by a remission instead of an intermission in the febrile paroxysms. It is rare in temperate climates, and is attended with large mortality.

Symptoms.—The *onset* is more abrupt than in intermittent fever. Prodromata are not so common, but when they occur they are of the same character. The chill is not usually so violent, nor the cold stage so long as in intermittent fever; on the other hand, nausea and vomiting are common, and in some cases there are bilious vomiting and diarrhea, tenderness over the stomach and spleen, and sometimes jaundice. The temperature rises rapidly from 103° to 106° F., and remains high for a longer time than in intermittent fever, the hot stage lasting in severe cases from six to eighteen or twenty hours. During this stage the patient suffers from headache, pains in the back and limbs, great thirst, and gastric irritability. A remission now succeeds. The temperature falls two or three degrees, but not to normal; free sweating occurs, the nausea and vomiting cease, and the patient becomes much more comfortable. In the course of some hours the temperature again rises, often to a higher point than before, but frequently without antecedent chill. Daily paroxysms usually occur, those on alternate days being severe. The temperature often reaches its highest point at the third paroxysm. The disease generally runs its course in from nine to twelve days, but it may last much longer. The type of fever may change to intermittent, which is a favorable sign, or become continued and again remittent, or remain remittent throughout; finally, the fever may subside gradually, or less commonly, by crisis.

Pernicious Malarial Fever.—Any of the malarial plasmodia may cause pernicious symptoms, but estivo-autumnal form is the causative organism in the great majority of cases. Numerous clinical manifestations of pernicious malaria are recognized, of which, however, (1) the *algid*, (2) the *comatose*, (3) and the *gastro-enteric* forms are the most common. The pernicious symptoms may develop after repeated paroxysms, or the first seizure may be fatal.

1. The *algid form* occurs more frequently than any other in this country. The patient is extremely weak, the entire body is cold, with

pinched features, blue lips, and faint voice. Respiration is shallow; the pulse rather slow, feeble, and irregular. There may be copious perspiration, but the internal temperature is very high. Deep stupor may be present. Unless speedy relief can be afforded the attack ends fatally.

2. In the *comatose form* the patient is completely unconscious, the skin hot and of a muddy, semijaundiced hue. Both pulse and temperature are increased. In other cases coma is preceded by wild delirium resembling acute meningitis.

3. The *gastro-enteric form* has for its prominent symptoms nausea, vomiting, diarrhea, intense thirst, extreme restlessness, a frequent, feeble pulse, and urgent dyspnea. The patient is cold and partly collapsed. Reaction may or may not occur.

Sequelæ.—Neuritis, paralyses, psychic disturbances, nephritis, and rupture of the spleen occur at times. A postmalarial secondary anemia is a frequent sequelæ.

Malarial Cachexia occurs especially in those who have had repeated attacks of malaria. The patient suffers from dyspepsia and constipation, with occasional bilious attacks; the face is of a pale lemon-yellow color, and may be slightly jaundiced; there is marked anemia, together with great enlargement of the spleen (ague cake) and some enlargement of the liver. The patient is weak and languid, and sometimes has considerable mental depression. Hemorrhages are common and fever variable.

Laboratory Diagnosis.—A positive diagnosis of malaria can only be made by finding the parasites in the blood so that the blood examination in suspected cases is absolutely essential. (See page 363.) There is an anemia of hemoglobin and red blood cells, most pronounced in cases of long standing. A leukopenia is present with a relative increase of large mononuclear cells. Pigment is frequently found in the white cells. Albuminuria is present in from 50 to 60 per cent. of the cases. Hemoglobinuria is recognized by some as a specific disease, by others as a complication of malaria.

Diagnosis.—The essential points in the diagnosis of intermittent fever are the periodical recurrence of paroxysms of chill, fever, and sweating, associated with the presence in the blood of pigment and parasites, and with enlargement of the spleen. The so-called therapeutic diagnosis may be made—an intermittent fever which does not yield to proper doses of quinine is not malarial. A typical malarial intermittent fever is not likely to be mistaken for anything else. (See Fever, pages 212 to 220.) It needs, however, to be distinguished from *other intermittent fevers*. Such fevers occur in tuberculosis, in the puerperal state, in empyema, subphrenic abscess, abscess of the liver, or, indeed, in any form of suppuration. Here, also, there are recurring chills, with fever and sweating, but the attacks are not regularly periodical and intermittent; sometimes the fever is intermittent and sometimes remittent; the chills recur at irregular intervals

and are not so violent as in the malarial attack. The essential difference, however, lies in the fact that a local cause can be found to explain the fever.

The intermittent fever of *hepatic* origin is not regularly intermittent, is not controlled by quinine, and jaundice may be present.

Urethral fever, may be mistaken for malarial fever; but the paroxysm is usually single, and the history of the operation and the absence of plasmodia from the blood clear up the diagnosis.

Typhoid fever is distinguished from remittent malarial fever by its gradual onset, the absence of chills and vomiting, as a rule; and, on the other hand, the presence of epistaxis, delirium, rose-colored spots, tympanites and diarrhea, with pale yellow, watery stools. The temperature in typhoid is more continuously high, the daily oscillations being of shorter range. A history of exposure to malarial infection and of previous attacks can often be obtained. The results of the blood examination (Widal test) and the bacteriological studies would settle the diagnosis in doubtful cases. It must be remembered that a mixed infection occurs sometimes, in consequence of which the plasmodium of malaria may be found either at the beginning or in the decline of the typhoid infection.

Amebic Dysentery.—An infectious disease, caused by the *Entamæba dysenteriae* (*Entamæba histolytica*), running an irregular course and characterized by attacks of diarrhea and the finding of the amebas in the stools.

Symptoms.—The onset may be abrupt or gradual, with symptoms of intestinal catarrh. In most of the cases a frequent and painless diarrhea follows a period of slight ill-health. The diarrhea alternates with short periods of constipation; the stools are watery and contain mucus, but no blood. Occasionally the onset is sudden and the symptoms much more pronounced. The course of the disease is irregular. There may be intermissions and exacerbations of the diarrhea without obvious cause. Amebic dysentery may rapidly pass from a mild to a severe course, or become chronic. One form is the gangrenous, which may be unsuspected until autopsy shows it to have been present. True relapses are common, and the tendency to chronicity is great.

The *milder* cases are attended by weakness, emaciation, and pallor; the expression is dull; the skin is dry and sallow; the tongue pale, flabby, moist, and slightly furred; the abdomen is normal or retracted; the temperature does not rise above 100° F., and the pulse-rate ranges from 70 to 90 per minute.

In the *grave form* the face is drawn and the expression anxious; the mind is clear; anorexia, intense thirst, and sleeplessness are present. The abdomen is greatly retracted, and there may be free sweating. The temperature is normal or subnormal, the pulse small and rapid. Progressive anemia and loss of flesh are prominent and dominate the intestinal symptoms. The skin is dry and harsh, and of a dull, greenish-yellow color if the cases are protracted.

Diarrhea.—This may be the only feature of the disease. It is characterized by great variation in character and frequency in all grades and during different periods of the disease. Intermissions and exacerbations may be observed at any time. The latter begin suddenly, and subside in the same manner. They last from two to ten days. The intermissions continue from one day to three weeks, during which the feces are soft.

In the gangrenous form the stools number thirty or forty in the twenty-four hours at first, then decrease, so that toward the end of fatal cases they are reduced to three or four.

In the more moderate types if the attack is abrupt, the stools at the outset are copious, bloody, and contain the amebas; if gradual, the stools are fecal, liquid, containing mucus and streaks of blood, and many gelatinous grayish masses. Stools of this character number from four to ten in twenty-four hours, and may continue thus for weeks. In chronic dysentery there is not so much mucus or blood, except in exacerbations. The stools are of the consistence of thin gruel and have an earthy or dull yellow color. Mucus is persistently present, however, in the intermissions, when the stools are soft and fecal.

Colicky, abdominal *pain* is constant; it occurs in the early stages of both forms and in acute exacerbations. As the movements diminish, the pain decreases. In chronic cases the colic is complained of during the exacerbations; during the intervals there is a dull, aching or burning pain in the upper quadrants. Moderate tenderness on pressure is present in most cases along some part of the course of the large bowel.

In amebic dysentery, *fever* is not a prominent feature, although there is usually a moderate rise in temperature.

Chronic dysentery is afebrile. In exacerbations of diarrhea slight fever may occur. Complications cause a higher temperature.

Complications.—The complications of amebic dysentery are (1) hepatic abscess (*q. v.*), which may develop at any period of the disease; (2) hepatopulmonary abscess (see Abscess of the Lungs), occurring secondary to the liver abscess; (3) general peritonitis from perforation, an uncommon complication of amebic dysentery, but taking place occasionally in the gangrenous form; (4) local peritonitis without perforation, a rather common complication; (5) hemorrhage from the bowel, which may be sufficiently profuse to cause death.

Laboratory Diagnosis.—The characteristic findings are secondary anemia (pronounced in chronic dysentery), polynuclear neutrophilic leukocytosis, and amebas in the stools (see Feces, Parasites). The amebas may also be found in the fluid from exploratory paracentesis of the liver or in the sputum if complicating abscesses occur.

The urine is concentrated if the diarrhea is severe, and frequently contains albumin and casts.

Diagnosis.—An absolute diagnosis is achieved by finding the amebas in the stools. In any case of diarrhea accompanied by tormina and tenes-

SPECIAL DIAGNOSIS

mus and the passage of bloody stools the latter should be thoroughly searched for amebas.

Trypanosomiasis (*Sleeping Sickness*).—A chronic disease, caused by the *Trypanosoma gambiense*, the intermediate host of which is the tsetse fly, characterized by two stages, with a long period of latency between the two. The disease is prevalent upon the west coast of Africa and only occasionally found elsewhere.

Symptoms.—First Stage.—*Trypanosomiasis*.—This stage is characterized by general enlargement of the lymphatic glands. The trypanosomes may be demonstrated in the blood and lymph glands.

Second Stage.—*Sleeping Sickness*.—After a period of latency extending over months or years there develops mental hebetude, headache, weakness, intermittent fever, tremor of the hands and thick speech. The lethargy slowly increases and after many weeks the patient is unable to walk or speak. He lies in an absolutely comatose condition, emaciated and without control of the vesical or rectal sphincters, to die eventually in a state of coma.

Laboratory Diagnosis.—An absolute diagnosis is made by the discovery of trypanosomes in the fluid removed from a lymph gland with a hypodermic needle and syringe, in the blood, or in the cerebrospinal fluid. A lymphocytic leukocytosis is present.

Leishmaniasis.—*Kala Azar* (*Dum-dum Fever, Tropical Splenomegaly*).—A chronic, usually fatal Eastern disease, due to the *Leishmania donovani* parasite, characterized by irregular fever, enlargement of the spleen and anemia.

Symptoms.—The onset is extremely slow. The gradual enlargement of the spleen is first noticed, but finally it reaches an immense size, and irregular fever of all types develop, associated with subcutaneous hemorrhages, or hemorrhages from mucous membrane, and with chlorotic anemia, severe intestinal symptoms, transitory edema, and dyspnea. Secondary infection of all kind are common. The mortality is extremely high.

Laboratory Diagnosis.—The parasites, probably a stage in the development of a trypanosome, is found by splenic puncture. With the anemia there is a leukopenia with a relative increase in the mononuclear leukocytes.

Other Forms.—*Infantile kala azar* is the infantile splenic anemia seen in the countries bordering on the Mediterranean.

Tropical Ulcer (*Delhi Boil, Nile Sore, etc.*) is a chronic granuloma occurring in certain subtropical and tropical countries, characterized by formation of nodules in the exposed parts of the body which may or may not form indolent ulcerations. Both of these two conditions are caused by protozoa of the *Leishmania donovani* type, but differing somewhat from them in cultural and morphological characteristics.

Disease Due to Infusoria.—The most important infection is that of the *Balantidium coli*, which has been shown to be a cause of a chronic form of dysentery in the Philippines.

II. DISEASES DUE TO METAZOA

A. **Diseases Due to Flukes.**—**Distomiasis.**—The fluke infestations may be due to any one of a number of parasitic trematodes (see page 435). The organs usually invaded are the lungs, liver, intestines, and blood.

1. **Pulmonary Distomiasis** (*Paragonimus westermani*).—Hemoptysis and cough are the two characteristic symptoms. The sputum is yellow to rusty brown, due to the myriads of tiny eggs which are frequently mixed with blood. If the eggs enter the brain, Jacksonian epilepsy will develop.

2. **Hepatic Distomiasis** (*Fasciola hepatica*, *Dicrocoelium lanceolatum*, *Opisthorchis sinensis*).—Irregular and at times bloody diarrhea, enlarged and painful liver, irregular jaundice and ascites are the common clinical manifestations. The diagnosis is based upon the discovery of the parasitic eggs in the feces.

3. **Intestinal Distomiasis** (*Fasciolopsis buski*).—This clinical manifestation of distomatosis is exceedingly rare.

4. **Venal Distomiasis.**—**Bilharziasis** (*Schistosoma hæmatobium*).—The infestation is essentially chronic, death usually resulting from some intercurrent disease. There are two clinical manifestations of the disease, according as the ova are deposited chiefly in the lower genito-urinary tract or the rectum. The first form is characterized by hematuria (the blood appearing at the end of micturition), frequency of urination, lumbar and suprapubic pain, often going on to a severe vesical catarrh, urethral stricture, and later formation of urinary fistulæ. The second form is manifested by blood, mucous stools, diarrhea, tenesmus, and later, prolapse of the rectum.

In advanced or severe cases the patient becomes anemic, debilitated, and liable to some intercurrent infection.

Laboratory Diagnosis.—The spined ova are found in the urine or feces of the sufferer. At times in chronic cases it may be either necessary to scratch the bladder surface with a sound or break up one of the small, soft rectal growths in order to demonstrate the ova.

B. **Diseases Due to Cestodes.**—The habitat of tapeworms is in the intestines, although occasionally the larvæ infest various organs of the body.

1. **Intestinal Cestodia.**—The *Tænia solium*, *Tænia saginata*, and *Dibothriocephalus latus* are the tapeworms found most commonly inhabiting the intestinal tract of humans; less frequently the *Hymenolepis nana* and *diminuta* and the *Tænia cucumerina* (see page 434).

SYMPTOMS.—The symptoms persist as long as the parasite remains in the intestines. They are both local and general.

Local Symptoms.—Abdominal distention, vague pains, diarrhea, and itching about the anus may be local complaints.

General Symptoms.—Fetid breath and ravenous appetite are common. A severe anemia frequently develops, particularly with diboth-

riocephalus infections. Nervous symptoms of mental depression, hypochondria, convulsions, and vertiginous attacks indirectly develop at times.

LABORATORY DIAGNOSIS.—The discovery of the ova or segments of the parasites in the stools renders the diagnosis positive.

2. **Visceral Cestodia.**—Although infestation by the larvæ of the *Tænia saginata* and the *Cysticercus cellulosæ* is extremely rare, they may enter by way of the stomach any organ of the body. More frequently, however, they appear in the subcutaneous tissues as ovoid, whitish nodules. A positive diagnosis is only obtained by microscopic examination of nodules excised from the skin or muscles; rarely, when infestation of the eye exists, they are diagnosticated by the ophthalmoscope.

The larvæ of the *Tænia echinococcus*, *echinococci*, lodge most frequently in the liver or other abdominal viscera or the lungs, usually affecting but one organ. Here the eggs multiply to form large collections of bladder worms, with development of a hydatid cyst. (See Hydatid Disease of the Lungs, page 602, and Hydatid Disease of the Liver, page 708.)

C. **Diseases Due to Nematodes.**—The most important round-worm infestations are intestinal, muscular, and subcutaneous. (See pages 436 to 437.)

1. **Ascariasis.**—The *Ascaris lumbricoides* is frequently present in the intestines of young children and causes no symptoms as a rule, except some mild irregular gastro-intestinal manifestations. The severe symptoms that they may cause are the result of the migration of the worms from the intestines into the bile or pancreatic ducts, and into the stomach, when they may pass into the trachea, bronchi, or into the nasopharynx. They have been known to get into the bladder and to be passed during micturition.

2. **Oxyuriasis.**—The *Oxyuris vermicularis* (seat-worm) inhabits the lower intestines in large numbers and from there wanders out into the cutaneous folds in the region of the anus, causing terrific itching and extreme discomfort. Commonly affecting children, it is a frequent cause of restlessness, disturbed sleep, and anorexia.

3. **Trichocephaliasis.**—The *Trichocephalus dispar* (*T. trichiuris*) occurs in large numbers in the large intestine of the affected person, usually in conjunction with other intestinal parasites. They rarely cause symptoms.

4. **Trichiniasis.**—Invasion by the *Trichinella spiralis* is characterized by fever; gastric and intestinal irritation, followed by pain and stiffness in the voluntary muscles; edema of the eyelids, face, and feet; by profuse sweating, and by death or tardy convalescence. The larvæ enter the body through ingestion of improperly cooked or raw, infested pork. In the intestinal canal the embryos develop into worms, fertilization occurs here, and the resulting embryos pierce the intestinal wall and are carried by the blood or lymph to the muscles, where they become encysted.

Symptoms.—Swallowing of trichinous flesh does not necessarily produce symptoms. When symptoms result, the severity depends upon the number of trichinae that become liberated. The symptoms are sleeplessness, lassitude, anorexia, nausea, vomiting, tenderness over the abdomen, colic and diarrhea. Headache is a constant and marked symptom of invasion. Toward the end of a week some of the voluntary muscles become stiff, painful, and contracted, and feel hard and swollen. Depending upon the muscles involved, there are interferences with the eye movements, contractions of the jaw muscles, difficulty in breathing or in swallowing, etc. The calves of the legs are especially involved. Recurrent edema over the affected muscles, eyelids, and face is very common and characteristic. The fever is usually moderate, but it may be high, and either of the remittent or intermittent type.

Profuse sweating and itching are common. The later stages in fatal cases are marked by insomnia, delirium, stupor, and coma.

The *duration* varies from a few days to four or five weeks, or even longer. Muscular pains may persist for months after recovery. Death results from exhaustion, or from some complication, as pneumonia, or ulceration of the large intestine.

Laboratory Diagnosis.—A most marked absolute eosinophilia develops in trichinosis as well as an increase in the total number of leukocytes. In doubtful cases excision of a small piece of an affected muscle, in which the trichinella embryos can readily be demonstrated, is a simple procedure. The trichinellae are occasionally found in the blood.

Diagnosis.—The diagnosis is based upon the history, the peculiar muscular pains and swellings, the localization of the edema, and the leukocytosis and eosinophilia. The muscles are swollen and hard, painful on pressure, and contracted.

The resemblance of trichinosis to typhoid fever and muscular rheumatism is slight, and although possible, mistakes in the diagnosis are most improbable if the blood is examined.

5. Uncinariasis (Hook-worm Disease).—The *Uncinaria duodenalis* and *Uncinaria americana* of this family are two of the most dangerous and most prevalent of all intestinal parasites. The two parasites vary somewhat in morphology but present the same general characteristics. They are found universally in tropical and subtropical countries and in many mines in temperature climates. In this country the uncinaria has been the cause of great economic loss in the Southern States.

Symptoms.—The symptoms are due to the prolonged, persistent loss of small quantities of blood. The secondary anemia is the most characteristic result of the infestation and with it appear the usual symptoms of anemia. The pallid, waxy face, the heavy eyes, the pigmented, dirty-looking skin, the emaciated appearance, the mental hebetude and the physical weakness of the sufferer are most characteristic.

In advanced cases there develop enlargement of the liver and spleen and edema of the face, feet, and ankles.

Laboratory Diagnosis.—The ova are found in the stools of infested patients. The parasites may be recovered after an appropriate anthelmintic. The blood shows a pronounced diminution of hemoglobin and a reduction of erythrocytes. Leukocytosis is rarely present. An eosinophilia is present only in the moderate and light infestations.

6. *Filariasis.*—Infestation by the *Filaria bancrofti* (see page 365) is frequent among the natives of tropical and subtropical countries. The parasites are conveyed by the female mosquito of both the anopheles and culex family. The larvæ enter the blood stream and then make their way into the lymphatics. Here they reach their sexual maturity and give birth to new larvæ which enter the blood and lymph in large numbers.

Symptoms.—*Filaria* may be present in the blood for a long time without causing any symptoms. After a while, however, enlargement of the spleen, anemia, and irregular fever develop. When blocking of the lymph channels occurs, hematochyluria, lymph-scrotum, varicose inguinal glands, and elephantiasis of the extremities are the most frequent secondary results.

The hematochyluria is intermittent and usually is the result of rupture of a lymph-varix in the bladder wall. In lymph-scrotum and elephantiasis the scrotum and extremities slowly attain a large, often enormous, size. The varicose inguinal glands are soft, doughy enlargements, somewhat resembling a hernia.

Laboratory Diagnosis.—The filaria embryos (*Filaria nocturna*) are found to be most numerous in the blood at night. There is secondary anemia usually, particularly if there are severe complications. The leukocytes are increased at first, later they return to normal. The urine may be milky, fatty, and readily coagulated from the chyle. Usually red-blood cells are mixed with the chyle.

Other forms of filaria are occasionally found in the blood. Of these the *Filaria perstans* and *F. diurna* alone are thought to be of pathological import. The former is said to be the cause of *craw-craw*, a papillo-pustular skin eruption of West Africa; the latter, found in the peripheral circulation during the day, is the larva of the *Filaria loa*, which inhabits the connective tissues all over the body and is thought to be the cause of the so-called "calabar swellings."

7. *Dracontiasis.*—A disease of Africa and the East Indies, occasionally seen in this country, caused by the *Dracunculus medinensis*, is characterized by the formation of vesicles and ulcers in the subcutaneous tissues. The parasites enter the intestine, but the female alone makes her way to the periphery, where the worm can be left at times and the head can be seen at the base of the ulcer when the vesicle breaks down.

8. *Trichocephalosis.*—The *Trichocephalus dispar* is usually found in association with other intestinal parasites. It has been considered to be non-pathogenic, but in a certain number of cases it is certainly the cause of enteritis, diarrhea, and severe anemia.

CHAPTER XXXIII

SUNSTROKE—THE INTOXICATIONS

SUNSTROKE

SUNSTROKE (siriasis, thermic fever) is apart from the infectious disorders the most pronounced expression of fever. The flushed face, the pungent skin, the pronounced dyspnea, and the extremely rapid pulse forebode the high body temperature, which in the axilla may reach 108° to 112° F. in a very short time. If recovery is the ultimate outcome the temperature moderates, but continues for a few days. In some instances dyspnea, heart failure, and coma may follow so rapidly that death ensues in one or two hours. In other cases pain in the head, dizziness, and languor precede the stupor. Nausea and vomiting, perhaps diarrhea, chest oppression, frequent micturition, and convulsions may precede insensibility. Unconsciousness, quickly or gradually lost, may be transient or pass into deep coma. Relaxation of the muscles with twitching is seen, and the pupils, at first dilated, become contracted.

The diagnosis is based on the history, the mode of onset, and the hyperpyrexia. Thermic fever must be distinguished from uremia and apoplexy (*q. v.*).

Heat Exhaustion is readily recognized. The moist, pale, and cold skin, the soft, feeble pulse, the quiet but hurried breathing, are unattended by fever. The shock is not attended by coma, and usually responds to treatment.

THE INTOXICATIONS

Toxins are of two types: *exogenous*, arising outside the system, and *endogenous*, arising within. *Intoxication* is the process by which toxins exert a deleterious action on the body cells.

Adami classifies the intoxications as follows:

I. **Exogenous Intoxications.**—1. *Non-parasitic.*—Intoxications not produced in the system but which gain entrance to the body through the skin, alimentary, respiratory, or urinary tracts.

2. *Parasitic.*—(a) Parasitic proper—due to introduction and growth within the tissues of animal and vegetable parasites, which elaborate toxins; (b) saprophytic—due to the growth of parasites on a surface in relation to the exterior of the organism, the products of the growth becoming absorbed and diffused into the tissues.

II. Endogenous Intoxications (autointoxication proper).—1. *Internal Secretory*.—Under this heading are grouped the disorders that arise as a result of alterations in the secretions of the glands of internal secretion. Thus disturbance of the thyroid secretion causes myxedema (deficient secretion) or exophthalmic goitre (hypersecretion). Removal of the parathyroids causes tetany. Alterations in the internal secretory functions of the pituitary and the pancreas cause various symptom-complexes. Disturbances of the other portions of the body which are without actual demonstrable secretory glandular structure are known to elaborate secretions (hormones) which seem to be necessary in order to excite to secretion closely related glandular structures. The syndromes produced by absence or perversions of the various hormones are as yet unknown.

2. *Non-eliminated Products of Katabolism*.—In this case intoxication may be brought about either by the failure of the excretory organs to eliminate toxic katabolic substances or by the resorption of excreted material from obstruction to the eliminative channels. In the first case uremia is an example of the result of failure of organs to perform their excretory function and in the second case obstructive jaundice, with its train of toxic symptoms, or acute pancreatitis, from obstruction of the ampulla of Vater, are typical examples of this form of intoxications.

3. *Disintegrative Intoxication*.—"Autolysis is the process of self-disintegration that tissues undergo." For example, in the liver, in cases of phosphorus-poisoning, acute yellow atrophy, chloroform-poisoning, and pernicious vomiting of pregnancy, the causative toxin probably destroys the cells without interfering with their ferments, which are still capable of causing autolysis, with resulting extensive necrosis of the liver parenchyma and consequent severe intoxication. Extensive burns may destroy the body cells with the production from the disintegrated cells themselves of a powerful toxin, which may cause overwhelming symptoms of intoxication.

4. *Impaired Metabolism*.—Failure to carry out the normal metabolic processes to their normal termination by the organism with the consequent discharge of toxic substances, and accumulation of non-toxic substances through deficient oxidation, causing alterations in the functions of the tissues, are important causes of autointoxication. Gout, obesity, alkaptonuria, hemochromatosis, rickets, or acidosis and acetonuria (occurring in diabetes, starvation, pernicious vomiting of pregnancy, etc.) may all be manifestations of endogenous intoxication arising as a result of metabolic disturbances.

III. Intoxications not Purely Exogenous or Endogenous.—Under this heading Adami classifies the gastro-intestinal intoxications that occur in constipation and intestinal obstruction. The general feeling at the present time, however, seems to be that the intoxication that arises as a result of constipation is a purely endogenous intoxication, arising as a result of abnormal fermentation with the resulting production of

toxic substances or as a result of the toxins elaborated by the bacteria themselves. This intoxication that arises as a result of the absorption of toxic products from the alimentary tract can in no way be considered an autointoxication or a true endogenous intoxication.

Chronic Intoxications.—The following conditions due to chronic, long-continued or slowly developing non-parasitic exogenous poisoning are relatively common. Other forms of such chronic intoxications have been omitted, as their occurrence is so extremely rare. The acute forms of poisoning have also been omitted, as they belong more properly to the subject of toxicology.

Opium Habit.—*Morphinism.*—The diagnosis is based on the history, when obtainable, on the evidence of malnutrition without cause, on the general depression and lassitude, and upon the temperament of the patient; to which symptoms are added insomnia, restlessness, and itching of the skin. A peculiar sallowness of the complexion and a prematurely aged appearance are also noted. Pseudoneuralgic pains are common, tabetic symptoms may be present, and notably gastrointestinal symptoms, as gastralgia, vomiting, and diarrhea (especially if the drug is withheld). Fever may be present, intermittent or continuous.

Cocaine Habit.—*Cocainism.*—Habitual users of this drug become sooner or later physical wrecks, and develop pronounced mental symptoms. The habitué, when not under the immediate effect of the drug, may be stupid, morose, and irritable, later developing hallucination of sight and hearing and other psychoses.

Alcoholism.—*Acute Intoxication.*—In this state the reeling gait and the incoherent speech, followed by narcosis, are generally familiar. Heavy breathing, a full pulse, dilated pupils, a stuporous rather than comatose state, are accompaniments of this intoxication. It is often necessary to differentiate acute alcoholism from uremia, apoplexy, fracture of the base of the skull or opium poisoning, and it is frequently a difficult problem. Furthermore, the possibility of any of these conditions occurring in a drunken subject must be borne in mind.

Chronic Alcoholism.—When alcohol is taken for a long time, tremor of the hands and tongue are seen and the action of the muscles is unsteady. The mind is dull, the temper irritable; forgetfulness is most common; later insanity, pseudoparesis, or epilepsy may ensue. Alcoholic polyneuritis is of frequent occurrence, and is often associated with mental confusion, disorientation of time, place, and person, and imaginary reminiscences and marked hallucinations (Korsakoff's syndrome). Gastro-intestinal catarrh, with poor appetite and constipation, is most liable to ensue, and later cirrhosis of the liver and kidneys; endarteritis with its train of pathological processes, including myocarditis and visceral scleroses, also arise.

Delirium Tremens (Mania a potu).—Prolonged acute alcoholic poisoning causes at times a psychosis characterized by terror-inspiring hallucinations of sight and hearing, restlessness, tremor, insomnia,

continuous muttering, rapid pulse and elevation of temperature, and marked prostration. The condition is frequently brought on by the suspension of alcohol after prolonged debauches and may develop as a result of shock or accident or during the course of an acute disease, especially pneumonia.

Lead-poisoning.—Intoxication due to lead, or plumbism, may be acute or chronic. It occurs chiefly in workingmen engaged in the manufacture or use of white lead, although cases of plumbism in those susceptible to the poison have been reported from a variety of causes.

The manifestations of chronic poisoning include one or more of the following conditions:

(a) *Saturnine cachexia*, in which anemia is most pronounced. The red cells show granular degeneration and polychromatophilia, extremely suggestive findings.

(b) *Colic*, usually preceded by indigestion and constipation, is often acute in onset and extremely severe, with the pain referred to the navel and at times accompanied by vomiting and diarrhea.

(c) *Paralysis*, which may be acute, subacute, or chronic, and which usually develops without fever. The paralysis may be antibrachial, causing characteristic wrist-drop; brachial, in which the scapulohumeral form of paralysis is seen, or of the Aran-Duchenne type, resembling chronic anterior poliomyelitis; and peroneal in type, in which the lateral peroneal muscles, the extensor communis of the toes and the extensor proprius of the big toe, are paralyzed, causing foot-drop and the steppage gait. Finally, paralysis of the abductor muscles of the larynx may occur in lead-poisoning. The paralysis often extends from a local group of muscles throughout the body, presenting symptoms like those of an ascending paralysis with rapid wasting. In other instances the general paralysis occurs primarily, the wasting and loss of power going hand-in-hand. Fever sometimes attends a general paralysis in lead-poisoning.

(d) *Cerebral* symptoms (encephalopathy) are usually rare, although optic neuritis or neuroretinitis is relatively common, as is tremor and headache. Delirium with hallucinations may occur as well as convulsions and epilepsy.

(e) Chronic lead-poisoning leads to *arterial sclerosis* and *contracted kidneys*, with hypertrophy of the heart.

(f) Chronic *gout* is frequently seen, but the joints usually involved are not the ones commonly affected in ordinary podagra.

(g) As described in the section in which the mouth and gums are discussed the *blue line* is the specific symptom of lead-poisoning.

Arsenic Poisoning.—Chronic arsenic poisoning may develop in those engaged in the manufacture of colored papers and its products, and colored dress goods. It may develop in users of arsenic-cured furs and at times follows the prolonged use of arsenic as a drug.

Various skin lesions, such as hyperidrosis, glossiness, pigmentation and keratosis, are the most common manifestations of chronic arsenic

poisoning. In addition there is often irritation of the mucous membranes of the mouth and pharynx, and there may be subacute gastro-intestinal catarrh, with diarrhea. In other instances there is profound anemia and debility, with paresthesia and neuralgia. In others, again, paralysis like that of lead palsy may occur. It must not be forgotten that puffiness under the eyelids may be due to this cause.

Mercury Poisoning.—Dye-workers, mercury-miners, hat-makers, and makers of fireworks are most frequently affected. The chronic type of the disease is characterized chiefly by ptyalism. There is seen in some cases, tremors, which ultimately may become extremely severe, and emotional disturbances often simulating hysteria, and which may develop into a true psychosis.

Phosphorus-poisoning.—This poisoning is seen chiefly in match-makers, and is characterized largely by necrosis of the jaw and its secondary results.

Silver-poisoning (*Argyria*).—Following prolonged administration of silver salts for therapeutic purposes, there develops a slaty-gray pigmentation of the skin, more pronounced in exposed parts.

Carbon-monoxide Poisoning.—Acute illuminating gas-poisoning is characterized by throbbing pain in the head, nausea, muscular weakness, and drowsiness, to be followed in severe cases with loss of consciousness and at times convulsions. The patient is cyanotic in appearance, but blood taken from the patient is a bright cherry-red color and gives the ordinary reactions for carbon monoxide. Pulmonary sequelæ are common. Chronic poisoning by repeated inhalations of CO is a frequent occurrence in the workers in municipal gas plants. Throbbing in the head, at times vertigo, physical weakness, a slow pulse, and usually a pronounced polycythemia, are the important symptoms.

Food Intoxications.—Among the intoxications which give rise to fever are those due to food-poisoning. Meat, milk products, and shellfish cause an intoxication which often threatens life, and from the suddenness of the attack, and the severity of the symptoms, suggests a bacterial infection rather than a food intoxication.

The history of the case is often the first clue to its nature. The symptoms are those of acute gastro-intestinal irritation, to which are added, with or without afebrile periods, the symptoms of collapse.

Meat-poisoning.—Poisoning by meat is due to bacterial (*B. botulinus*, *B. enteritidis*) infestation of the meat or to putrefaction, with poisoning from bacteria and the products of protein hydrolysis. Any animal food may cause the infection, but sausage, liver-pudding, ham, and other pork products are more frequently affected than other meats. The poisoning is followed by a period of incubation lasting from twelve to forty-eight hours, characterized by malaise, loss of appetite, nausea, and colicky pains. As these symptoms increase, chilliness ensues, and in some instances there is a marked rigor followed by a rise in temperature to 101° to 104° F. Prostration occurs almost immediately, with giddiness and faintness, and the occurrence of cold perspiration. Headache

and headache or delirium occur. Following the chilliness the symptoms of gastro-intestinal irritation arise, diarrhea being more frequent than vomiting. The abdominal pain increases and the perspiration and rising sweat become more pronounced. As further evidence of the intoxication, there is an extreme degree of muscular weakness. In addition to muscular weakness, cramps in the legs and arms, often followed by convulsive movements, occur, and the patient complains of paresthesia of various forms. In milder cases the symptoms of gastro-intestinal irritation and of muscular weakness alone attend the fever. In the more severe cases, the most pronounced symptoms are seen; the fever is replaced by a subnormal temperature and the patient presents all the other signs and symptoms of severe shock.

Poisoning by Milk and its Products.—As milk is a splendid culture media, and as it is usually ingested uncooked, poisoning by it or its products is relatively common. The germs of infectious diseases (typhoid fever, diphtheria, tetanus, scarlatina) may be conveyed by impure milk, but more properly speaking milk-poisoning, or poisoning by its products (cheese, butter, ice-cream, etc.), is due to the common forms of milk bacteria and their toxins. Such poisoning is characterized by severe gastro-intestinal symptoms. Many of the diarrheas of childhood and infancy are types of this intoxication, and are accompanied by a high degree of fever. In cheese-poisoning the fever is not continuous as it is in the other forms, and shock develops with greater frequency.

Poisoning by Shell-fish.—In mussel-poisoning, the symptoms are those of an acute neuro-poisoning, with profound nervous symptoms. Fever does not attend this condition, but collapse follows quickly, usually without gastro-intestinal symptoms. Poisoning by lobster or crabs causes similar symptoms. Fish-poisoning (scombrism) is also characterized by fever, collapse occurring early and at times associated with severe gastro-intestinal symptoms.

Grain-poisoning.—*Ergotism.*—The prolonged eating of a parasitic fungus (*erga*—*Claviceps purpurea*) which grows in the flower of certain European grains, notably rye, causes two forms of intoxication:

The *gangrenous form* is characterized by pain or anesthesia, tingling and muscular spasms in the small extremities, which is followed by gangrene of these parts.

The *convulsive nervous form* is chiefly manifested by headache, muscular spasms or cramps and moderate fever; mania, melancholia, dementia, or epilepsy may develop later. Tabetic symptoms are common.

Lathyrism.—The presence of the powdered seeds of the *Lathyrus sativus* in grain causes symptoms resembling spastic paralysis which may proceed to paraplegia. The condition occurs in the Mediterranean countries and in India.

Pellagra.—A chronic disorder characterized largely by gastro-intestinal and cerebral and cutaneous manifestations. The cause of the disease

is unknown, but the present belief seems to be that it is due to some unknown animal parasite, conveyed by the Simulian fly. However, the theory that it is due to the ingestion of diseased corn, or the fungus which infests mouldy corn, has been by no means abandoned and the United States Government Pellagra Commission has recently shown that this diet (corn) is in some way connected with the disorder, whether it is the actual cause of it or simply predisposes to it. The disease at first thought to be a European disease entirely, has been found to be very extensive in our Southern States as well as occurring less frequently elsewhere in the United States.

Symptoms.—The disease is insidious in onset and usually begins in the spring of the year as an erythema, which is succeeded by a scaly, wrinkled condition of the skin, particularly of the air-exposed parts. Crusts may form beneath, in which pus may be found. Along with the cutaneous manifestations, digestive disturbances of all kinds may be noted, of which salivation and intestinal derangements are most marked. In the more severe, graver cases, or in long-continued cases, nervous symptoms make their appearance as spasms, paralysis of the lower extremities, convulsions, impairment of mentality, melancholia and mania. On account of the cerebral disturbances, many cases unrecognized as pellagra have been confined to insane asylums throughout the country.

As the condition progresses in the severe cases, the strength is lost, the patient becomes gravely anemic, and usually dies in terminal cachexia.

Beriberi.—A disorder with fever which prevails in endemic form in tropical and subtropical countries, characterized by multiple neuritis associated with anasarca. By many observers it is believed to be an acute infection. Conditions predisposing to infections, such as overcrowding, hot and moist weather, and exposure to the elements, are usually present. It frequently is an institutional disease. It is far more common in men, and usually attacks subjects between the ages of fifteen and thirty. The disease is also believed to be a disorder of metabolism, instigated by absence of some unknown element in the food. The exact relationship between food and beriberi is unknown, but the food question has certainly a direct bearing on the etiology of beriberi, which may be explained on the ground that the rice diet of Eastern countries predisposes to the disorder or that the pericarp of rice contains certain materials that are necessary for the human economy as shown by Schaumann; the milled rice of the East has had the pericarp removed.

Symptoms.—Several clinical forms are seen. In the most complete form there is rapid loss of power in the legs and arms, with atrophy of the muscles. The patients complain of pain, and later, edematous symptoms may appear. With the loss of power in the legs there is paresthesia, with frequent palpitation of the heart and dyspnea. The pain in the muscles is associated with weakness and tenderness. In milder

degrees of this form, pain, weakness, in the legs, diminution of sensibility, and paresthesia are the most common symptoms. The onset is gradual, and the symptoms may recur from time to time, are much aggravated during the warm season.

Following the pain and weakness of the muscles, in some cases edema becomes very pronounced, associated with effusions into the serous cavities. In this so-called wet or dropsical form atrophy of the muscles is not observed until the edema disappears. In some instances the infection is very intense, and is characterized by more marked cardiac symptoms. In these instances acute dilatation may be followed by cardiac paralysis and death in twenty-four to forty-eight hours.

The diagnosis is based upon the occurrence epidemically or endemically in tropical regions of peripheral neuritis with edema. Thus the blood-picture has not been satisfactorily studied.

CHAPTER XXXIV

METABOLIC DISEASES

Gout.—A chronic disorder of purin metabolism characterized by attacks of acute arthritis, associated with excess of uric acid in the blood and the deposit of monosodium urate in and around the joints.

Etiology.—The pathogenesis of gout is unknown, but the following theories of the causation of gout are generally accepted. The blood in gout contains an excess of uric acid which is variously attributed to retarded formation of uric acid from nucleic acids, to diminished oxidation or destruction of the uric acid, and to diminished excretion by the kidneys. One or the other of these factors results in the accumulation in the blood of an excess of monosodium urate. The prolonged retention of the monosodium urate in the blood renders it more stable and less soluble, so that it is more likely to be deposited in the tissues, causing the familiar gouty tophi. The acute attack may be brought on either by an oversaturation of the blood with, and consequent rapid deposition of, the monosodium urate in the joint (just before and just after an acute attack there is a low excretion of acid, during the attack an excessive excretion), or by the absorption of the urates, previously deposited in the joint, resulting in inflammation of the neighboring lymphatics and excessive excretion of uric acid.

Gout is common in Europe, particularly in England, but in its articular form is rare in this country. There is an hereditary predisposition in from 50 to 60 per cent. of the cases. It results from overeating of rich foods and the drinking of malt liquors, associated with insufficient exercise and excretion. Garrod has called attention to its association with lead-poisoning. Paroxysms are induced by indiscretions in eating or drinking, by nervous shock or great mental strain, by exposure to cold or injury, or by overwork and sexual excesses.

Acute Articular Gout.—The characteristic phenomena of acute gout are often preceded for a variable time by acid flatulent dyspepsia, colicky pains in the stomach and bowel, constipation alternating with diarrhea, and the excretion of scanty, acid urine. Accompanying these dyspeptic symptoms often are impairment of physical and mental vigor, irritability of temper, and hypochondriasis. In other cases the premonitory symptoms are palpitation of the heart, or dyspnea resembling asthma, or various nervous symptoms, as drowsiness, insomnia, or headache. However, the onset is often sudden, especially in the first attack. The ball of the great toe is hot, swollen, red, and exquisitely resentful to the slightest touch or jar of the bed.

The veins are swollen and the joint stiff. There are slight fever, perhaps chilliness, thirst, coated tongue, constipation; scanty, high-colored urine depositing urates on cooling; the skin is warmer than normal, and there is slight perspiration. The pain usually abates during the day and increases at night. It is aggravated by motion and attended by painful muscular cramps. By the end of the first day or two the swelling increases and the pain lessens, owing to diminished tension of the part. Pain on motion is still great, however, and without treatment may continue for a week or two. Both great toes may be attacked in the first seizure, more often alternately than simultaneously, and sometimes other joints than those of the toe are affected. A second attack may be postponed for several years, but usually after that the intervals of freedom steadily diminish until an attack recurs every few weeks or months, and the patient may be scarcely ever free from it. Other joints than the toes, particularly those of the fingers, become involved in subsequent attacks. During the attacks there is a hyperleukocytosis and a transient albuminuria if the kidneys are not already affected.

Chronic Gout results from repeated acute attacks. It is characterized by deformity of the affected joints, around which are deposited chalk-stones (tophi) of monosodium urate. Similar deposits occur in the helix of the ear. The first appearance is that of a clear vesicle under the skin, which subsequently becomes chalky-white and solid. At times the tophi ulcerate through the overlying skin. The deposits of monosodium urate occur not only in the cartilages of the joints, but in the ligaments and bursæ also, resulting in great impairment of motion and deformity. Arteriosclerosis with its symptoms and sequelæ gradually develop, so that as a result of repeated attacks in long-standing cases the patient may present the signs and symptoms of chronic interstitial nephritis, myocarditis, cerebral sclerosis, etc.

In so-called *retrocedent gout* the external joint-manifestations disappear rapidly, and synchronously there develops severe symptoms, referable to the internal organs. Thus there may be severe abdominal pain, vomiting, diarrhea, weakness and rapid pulse, or there may be precordial pain, dyspnea, and palpitation, or mental excitement, delirium, and coma. *Irregular gout* is a term applied to a variety of conditions of which but little is known, so that the term expresses ignorance more than anything else.

Arthritis Deformans.—Arthritis deformans is an affection characterized by acute or chronic inflammation of the joints, of progressive character, and resulting in deformities.

Etiology.—The etiology of the disease is unknown. It is variously attributed to (1) a disturbance of metabolism; (2) a trophoneurosis; (3) a toxemia; (4) an infection. The present idea seems to be that it is entirely due, indirectly or directly, to bacterial agents, whether the result of the absorption of the toxins elaborated by bacteria at some localized site of infection or the result of a systemic infection.

The acute exacerbations that occur in the course of the disease are certainly highly suggestive of a systemic infection, as is the frequent finding of some hidden, unsuspected focus of infection (chronic tonsillitis, pyorrhea alveolaris, sinusitis, genital and gastro-intestinal infection).

Clinical Varieties.—All forms of chronic so-called idiopathic joint lesions to which the terms rheumatoid arthritis, rheumatic gout, chronic articular rheumatism, etc., have been erroneously applied, are considered as varieties of arthritis deformans. Fitcher classifies the disease under three heads: (1) atrophic arthritis; (2) hypertrophic arthritis; (3) chronic villous arthritis.

1. *Atrophic Arthritis.*—This variety is a polyarthritic disorder that first attacks the small joints of the hands and later the small joints of the feet, the wrists, elbows, shoulders, ankles, knees, the

FIG. 178



Arthritis deformans. (Original.)

temporomaxillary and other joints. The disease is essentially chronic, but it may commence acutely with local and constitutional symptoms, closely resembling acute articular rheumatism, which pass off leaving the joint slightly deformed. Repeated attacks occur at irregular intervals followed by greater deformity. Ultimately extreme deformities ensue from atrophy of the articular surfaces, from ankylosis of the joints, and from pronounced wasting of the muscles around the joints, though the stronger muscle groups first overcome the weaker, causing subluxations, lateral deformities, flexions, or extensions. In another series of cases the condition is apparently insidious in onset, the joints becoming more and more deformed. With this there is a certain amount of pain and slight swelling around the joints. Acute exacerbations may occur at times, and again the progress is interrupted from time to time by omissions. The disease is steadily progressive in

most cases, though at times the condition may cease or become quiescent.

Other symptoms that frequently attend this variety of the disorder are general malnutrition, rapidity of the pulse, numbness and tingling around the joints, and a peculiar softness of the overlying skin, particularly noticeable in the palms of the hands, which are often moist and clammy at the same time.

Still has described a manifestation of the disease in children, characterized by a rather acute onset, some fever, multiple fusiform joint lesions, and enlargement of the spleen and lymph glands.

2. *Hypertrophic Arthritis*.—This type is characterized by hypertrophy of the cartilages along the outer margin of the joint, or at the attachment of the ligaments. This thickened cartilage eventually becomes ossified (osteophytes), causing more or less joint fixation. The smaller joints are usually affected and the condition is quite common in the fingers (Heberden's nodules and Haygarth's nodosities). Involving some of the vertebræ, the process causes marked rigidity of the affected portion of the body (spondylitis deformans). Affecting the larger joints (hip, knee, and shoulder) the condition gives rise to joint disability, causing the condition known as rhizomelic spondylosis. This hypertrophic form usually affects but one joint or a series of closely related joints, as the fingers or vertebræ.

3. *Chronic Villous Arthritis*.—This condition, usually affecting the knee-joint, is caused by villous outgrowths from the synovial membrane. It is characterized by some pain, stiffness, and marked crepitation of the joint. Usually only one or two joints are affected.

Diagnosis.—Arthritis deformans is distinguished from gout by the absence of an hereditary tendency. Arthritis deformans is progressive, with occasional remissions; gout occurs in successive attacks, with intermissions. Arthritis deformans in the vast majority of the cases is subacute or chronic. The acute form is distinguished from acute gout by the duration of the paroxysm and the absence of intermission; by there being less heat, swelling, and redness of the joints, and less infiltration of the soft parts; by the fact that large and small joints are involved, and that there is no special tendency to inflammation of the great toe.

From chronic gout, arthritis deformans is distinguished by the absence of hereditary predisposition, of repeated acute attacks, and of the causes of gouty paroxysms. Gout has a predilection for the great toe, and is unilateral. Gout attacks well-fed males, most frequently after the age of thirty years, while arthritis deformans tends to attack women. It may, however, occur in both sexes, and even be associated with gout.

Rheumatic fever is distinguished from acute arthritis deformans by its tendency to involve the larger joints, its erratic course, ac sweats, its shorter course, its tendency to heart complications, & its *subsidence without impairment of the usefulness of the joints*.

The joint-affections of *tabes dorsalis* are distinguished by the associated symptoms of incoördination and absent knee-jerk, by their sudden onset without pain or fever, by the occurrence of large effusions into the joints, with subsequent disorganization, fractures and dislocation.

Gonorrheal arthritis is distinguished by the history of gonorrhea or the existence of a discharge from the urethra, by the tendency of the disease to attack the larger joints, particularly the knee or shoulder, and to become fixed in one, not wandering from one to another. The affected joint suffers effusion, and the synovial membranes and bursæ are inflamed. The process is very chronic but indolent, and the heart rarely becomes affected.

Rickets (*Rachitis*).—A disease of infancy characterized by skeletal changes and general impairment of nutrition. The disease is probably produced by toxic influences, the result of metabolic changes due to improper nutrition, acting upon the skeleton.

Rickets usually develops, and is most common in children with unfavorable hygienic surroundings, who have lived upon a starchy diet and have taken cows' milk for too long a period of time. A child that has been nursed during the mother's pregnancy is apt to have the disease. With the faulty diet there is associated improper assimilation of lime salts.

Symptoms.—The onset is insidious, but gradually the child presents evidences of defective nutrition, such as marked pallor, flaccidity of the muscles, and general muscular weakness which results in an inaction resembling paralysis. Perspirations about the head are common, as there is usually more heat of the head than is natural. In sleep the child rolls the head, causing the hair on the back of the head to wear off. Late development of the teeth is observed. Later skeletal changes appear. If the ribs are examined, nodules will be detected at the junction of the bone with the cartilage, the *rachitic rosary*. The thorax is changed in shape. At the junction of the cartilages and ribs a depression takes place which is continuous with a groove passing out from the ensiform cartilage toward the axilla, *Harrison's groove*. It may deepen with inspiration. The sternum projects, forming the so-called "pigeon-breast." (See Thorax.) Changes at the lower end of the radius and ulna, and sometimes at the end of the humerus, are noticed. The parts are enlarged at the junction of the shaft and epiphyses. There may be thickening of the clavicles at the sternal ends. In the legs the lower end of the tibia becomes enlarged, and at times the upper end, or even the shaft, becomes thickened. The child becomes bow-legged, or the tibia and femora may arch forward. Knock-knee sometimes occurs. The bones of the vertebral column and of the pelvis are also affected. The spine is usually kyphotic, and sometimes there is lateral curvature as well. The contraction of the pelvis is such as to narrow its outlet—a matter of much importance for the future of females.

The child's head is quite characteristic. The fontanelles remain

open for a long time, and areas of ossification are imperfect, so that the bone yields to the pressure of the finger. This occurs particularly at the side, and the term *craniotabes* is applied to it. The large head is square in shape. The appearance of the face is peculiar; it is proportionately very small, especially in the lower two-thirds, while the forehead is broad and square.

The disease usually progresses slowly, and is eminently chronic. An acute form is seen at times. With some gastro-intestinal disturbances there are mild fever, considerable weakness, and great restlessness. Sleep is disturbed, and pain is complained of, if the child is of an age to make such complaint. Soreness of the body is observed on handling the child; and of its own accord, on account of the pain and soreness, it avoids all customary movements. The child lies on its back and shrinks from attempts to disturb it. Sometimes the most marked manifestations of the more acute forms are the gastro-intestinal symptoms.

In both the acute and the chronic forms, enlargement of the liver and spleen is observed. The enlargement is not only actual, but a false enlargement may also be seen from distortion of the organs on account of changes in the vertebræ and ribs. The abdomen is prominent, usually on account of flatulence, although the enlarged organs contribute to the swelling. *Rickety* children frequently suffer from tetany, *laryngismus stridulus*, convulsions, enuresis, etc.

Diagnosis.—The acute form of the disease must not be confounded with scurvy, as often happens in the case of children. In scurvy the pain, tenderness, and weakness are limited to the lower extremities. The gums are swollen and may be spongy or the seat of ecchymoses. The most decisive diagnostic criterion is the therapeutic test, scurvy rapidly yielding to a proper regimen.

Scurvy.—*Scorbutus*, or scurvy, is a chronic nutritional disorder brought about by a long-continued diet deficient in fresh vegetables, or in infants by artificial foods. It is characterized by pallor, great physical weakness and mental sluggishness, dyspnea, subcutaneous and submucous hemorrhages, a swollen, spongy condition of the gums, and a brawny induration, especially of the calves and hams, with discoloration of the overlying skin.

The joints are swollen, painful, and tender in about one-third of all cases of scurvy. The onset of the disease is usually gradual, and is marked by a peculiar dirty yellow or greenish pallor of the face, associated soon with an apathetic expression of the face, physical weakness, and decided lack of customary energy.

The gums swell almost always, become spongy, and bleed upon slight irritation. They are dark cherry red in color and sometimes they swell so as almost to hide the teeth completely. The breath has a heavy, sickening odor, and the teeth sometimes drop out of their sockets. In some cases the eye and its surroundings are the only parts exhibiting signs of scurvy at this time.

Constipation, rarely diarrhea and indigestion, is present and rheumatoid pains may be noted. Other prominent subjective symptoms are fatigue on slight exertion, dyspnea, faintness, and despondency. In the course of a week or two petechiæ appear upon the lower extremities. Depending upon the severity of the case there are also bullæ, vibices, and ecchymoses. Brawny induration, due to deep effusion of blood, occurs especially in the calves and hams, producing considerable pain on flexure of the knees and may break down to become foul ulcerations. There is no fever apart from complications.

In addition to the cutaneous and gingival bleeding, hemorrhages occur from the nose and other mucous surfaces, and effusions take place into the intestines, pericardium, and pleura.

A very peculiar symptoms, and sometimes the earliest, is hemeralopia, or night-blindness.

Infantile Scurvy (*Barlow's Disease*).—This is more or less common in infants fed on artificial food exclusively. It is therefore limited to the first four or five years. In fifteen cases I have seen, the most pronounced features were those of weakness, anemia, polyuria, restlessness, the scorbutic gums, subperiosteal hemorrhage about the epiphyses, particularly of the tibia, sometimes separation of the epiphyses, pseudo-paralysis of the legs, edema of the eyelids, petechiæ, and always a general tenderness of the body. In all of them the presence of a dietetic cause confirms the diagnosis.

Diabetes Insipidus.—The disease is often secondary to some organic disease of the central nervous system, frequently syphilis, and is then a symptom rather than a disease. The idiopathic type is probably due to a coexistent functional neurosis. In another type of the disease the kidneys seem functionally incapable of secreting a concentrated urine.

The disorder usually occurs in males, and is often hereditary. It is most common in young people. It may follow fright, a protracted debauch, or perturbation of the nervous system from other causes.

Symptoms.—This form of diabetes differs from the saccharine form in that the urine is normal except for its low specific gravity, from 1.001 to 1.005. The amount of urine may range from 5 to 20 liters. It is pale and watery. The solid constituents are unaffected as a rule. Urea is sometimes increased, but abnormal constituents are rare. The passage of large amounts of urine induces thirst. The patients are usually well-nourished, and have enormous appetites.

Diagnosis.—The diagnosis is not difficult. It must be distinguished from the polyuria that is seen in chronic interstitial nephritis, diabetes mellitus, and in amyloid disease. In hysteria, polyuria is common, although transitory. The presence of the stigmata and other hysterical manifestations establishes the diagnosis in hysteria.

Diabetes Mellitus.—Diabetes is a disorder due to the inability of the organism to properly metabolize carbohydrates.

Etiology.—The disease occurs in men more frequently than women (3 to 2). It is a disorder of all ages, but more commonly appears after

the fortieth year. It is a disease of the well-to-do and affluent, and often occurs in the same families for several generations. Diabetes is frequent among the Jewish race, rare in Negroes. Husband and wife are frequently affected (conjugal diabetes).

Prolonged toxemia from gross errors in eating (*e. g.*, uncooked or incompletely cooked starches, too rapid eating, too much food, and mental anxiety or stress while eating) are contributory causes (Hodgson). Conditions associated with transient glycosuria (*q. v.*) are frequently forerunners of true diabetes. The direct cause of the disease is probably some perversion of internal secretions, whether due to a failure of the internal secretion of the pancreas which inhibits sugar production by the liver, or to a hypersecretion of the adrenals and chromaffin system generally (from stimulation of the sympathetic system), the hypersecretion causing an overaction of the sugar-producing mechanism of the liver.

Symptoms.—The disease is characteristically insidious in onset and is frequently recognized by accident in routine examination of the urine, as in life insurance. The occurrence of one of the following conditions would suggest an examination of the urine—progressive muscular weakness without cause; rapid emaciation in young persons; excessive thirst; excessive hunger; and increased frequency of micturition, with polyuria.

The urine is of high specific gravity, pale, and contains sugar and frequently acetone. In a twenty-four-hour specimen the sugar varies from 0.5 to 6 and 8 per cent. in untreated cases (100 to 250 gms. sugar being excreted), in the treated cases the percentage and amount secreted varying with the severity of the disease and the diet. The total nitrogen is increased, from the normal 10 to 15 gms. per diem to 15 to 20 gms. or more. The ammonia output may be increased from the normal 0.5 to 1.5 gms. a day to 4 to 8 gms. and more, the ammonia excretion indicating the degree of acidosis present.

Complications.—The more important complications are referable to the nervous system and the gastro-intestinal tract. *Coma*, due to the accumulation in the tissues of organic acids from disturbance of fat metabolism (acid intoxication) may come on suddenly, may follow some infection, excitement, mental stress, etc., or may appear slowly, with weakness, headache, nausea, vomiting, and insomnia. As the condition advances the coma deepens and the patient cannot be aroused; Kussmaul's dyspnea appears, the pulse is rapid and weak, the pupils are dilated, and the temperature is subnormal. Death usually occurs within a few days. Among other nervous complications appearing from time to time in the course of the disease are neuralgia, peripheral neuritis, the so-called diabetic tabes, vague paresthesias, retinitis, herpes zoster, trophic ulcers, and epileptiform attacks. The gastro-intestinal complications include polyphagia or anorexia, vague symptoms of indigestion, constipation at times alternating with diarrhea. A dry, fissured tongue is frequently seen and the teeth readily dec

sponginess of the gums and gingivitis are frequent occurrences. The liver may be enlarged by itself or in conjunction with bronzing of the skin (hemochromatosis). The genito-urinary complications include nephritis, the urinary findings of which are particularly marked before the onset of coma; cystitis at times, associated with pneumaturia, vulvitis, vaginitis, urethritis, and balanitis. Amenorrhea is frequently noted and abortions are common. Loss of sexual power is frequently seen. General arteriosclerosis, with its train of symptoms and sequences, is common; gangrene of the lower extremities frequently develops as a result of it. Pulmonary complications are usually in the nature of terminal infections, the result of lessened resistance to bacterial infections. Local skin disorders, as boils and carbuncles, pruritus, eczema, and urticaria are common and annoying complications. The skin is harsh and dry and perspiration rarely occurs.

Diagnosis.—This depends upon the finding of grape sugar in the urine when the patient is upon an ordinary diet. If this persists the relatively uncommon transient glycosurias and alimentary glycosurias can be eliminated. However, in patients with apparently a transient glycosuria, the tolerance for glucose should be estimated before making such a diagnosis. 100 gms. of glucose are given upon an empty stomach. The patient voids urine at the end of six hours, preserving this specimen together with any urine voided before this, that is in the six hours after taking the sugar. The remaining urine, voided in the next eighteen hours, is also saved, but as a separate specimen. Tolerance is normal if no sugar appears. Alimentary glycosuria is present if there is a trace of sugar in the six-hour specimen and none afterward. Tolerance is subnormal if more than 2 gms. of sugar are excreted and if there is glycosuria in the eighteen-hour specimen (latent diabetes—Janeway). If a patient is found to have a true diabetic glycosuria it is of much importance to estimate the grade or severity of the case and to determine the presence or absence of acidosis, and if present, the degree. The severity of the case can be readily determined by placing the patient upon a protein-fat diet for three days. At the end of this time a known quantity of carbohydrate is added to the diet; for example, 90 gms. (3 ounces) of white wheat bread which contain 55 gms. of carbohydrate. In addition to this there will be a small quantity of carbohydrate in the cream given with the diet, so that the patient will take approximately 60 gms. of carbohydrate. The total quantity of the urine passed in twenty-four hours after the ingestion of the bread is measured and the amount of sugar excreted is estimated. If no sugar is found and there is no ketonuria the case is a mild one. Cases excreting a certain percentage of this sugar, and who show an acetonuria from time to time are moderately severe, and those in which more than the amount of carbohydrate ingested is excreted are severe cases. In the latter case there will always be disturbance of fat metabolism with persistent ketonuria. The degree of acidosis can be very roughly estimated by the degree of reaction of the qualitative tests for acetone

and diacetic acid, or by the amount of sodium bicarbonate necessary to neutralize the urine (norm. = 5—10 gms.), if there is no bladder or other local urinary tract complication. Reasonably exact estimations of the degree of acidosis are made by the estimation of the output of ammonia which is formed in increased amounts by the organism from its nitrogen in order to neutralize the excessive accumulation of acid.

Amyloidosis.—A metabolic disorder, developing in the course of prolonged suppuration.

Etiology.—The disorder is most common in advanced tuberculosis of the bones and in advanced syphilis, though any long-continued suppuration or non-suppurative cachectic condition may cause it. It occurs most frequently in young adults. The lesions are usually extensive, occurring most frequently, however, in the liver, spleen, kidney, and intestines.

Symptoms.—These are chiefly those of the preceding disease. Involvement of the liver occurs without jaundice, pain, or portal obstruction. The liver is uniformly enlarged, smooth, and with rounded border. At the same time the spleen is found to be palpable. Kidney involvement is suggested by the passage of large amounts of clear, albuminous urine, of low specific gravity, containing hyaline and waxy casts, and by the presence of edema. Intestinal involvement is shown by a severe intractable, painless diarrhea. In addition the general condition of the patient is typical. He becomes progressively anemic, weaker, emaciated, with pallid skin and with more or less anasarca.

Diagnosis.—The appearance of the above conditions, some more marked than others, in a patient who presents some etiological factor, renders the diagnosis unmistakable.

Obesity.—A metabolic disorder characterized by excessive fat development.

Etiology.—The condition is frequently hereditary, but in the greater percentage of cases the condition is actually caused by a secondary inactive life associated with overeating of fats and carbohydrates and overindulgence in alcohol.

Symptoms.—The condition is self-evident. Dyspnea, muscular weakness, impaired digestion, and constipation are the direct results of the disorder. Arteriosclerosis is frequently associated and myocardial insufficiency (fatty heart) is common.

Diagnosis.—Myxedema and edema are easily excluded. Several other forms of fatty accumulations are seen.

1. *Adiposis Dolorosa.*—(See page 141.)

2. *Adiposis Tuberosa Simplex* (Anders).—A condition characterized by fatty growths in the subcutaneous tissues of the extremities and the abdomen, somewhat resembling adiposis dolorosa, but amenable to dietetic treatment.

3. *Multiple Lipomatosis.*—Multiple, painless, circumscribed, fatty growths occur frequently without general obesity.

4. *Adenolipomatosis*.—Fatty, symmetrically distributed overgrowths in relation to the superficial lymph nodes, particularly of the neck, are seen at times.

5. *Syndromes of Fröhlich and of Cushing*.—The first is characterized by adiposity, smallness of stature and defective development of genital organs; the latter of adiposity, with overgrowths and sexual dystrophy. Both conditions are due to derangement of functions of the pituitary body, the adiposity resulting from deficiency of the posterior lobe secretion.

CHAPTER XXXV

DISEASES OF THE BLOOD AND DUCTLESS GLANDS

DISEASES OF THE BLOOD

General Considerations.—The blood is both a tissue and a fluid, and is in close relationship to all the organs and tissues of the body. It serves the functions of carrying to the body tissues oxygen and food and of removing from them the products of anabolism and katabolism. Furthermore, the blood contains the elements with which infectious and toxic diseases and conditions are fought.

Thus the blood is dependent upon many factors not necessarily directly concerned with it, but which may cause secondary changes in its constituents. Furthermore, the blood is directly dependent upon certain organs, the hemopoietic system, for the continual regeneration that is necessary to preserve its short-lived elements and alterations in the blood may be at times entirely due to functional changes in these organs; whether due to insufficient nutrition or actual disease. Lastly, alterations in the blood result in diminution in its functional capacity and the other organs suffer from the failure of the blood to bring nutrition and to carry off waste products. Thus the symptoms of blood affections are due to the effect of the altered blood upon the function or the nutrition of the organs. Many functional symptoms thus arising may be the first indications of blood disease, as vertigo, headache, dyspnea, or palpitation, all very common symptoms. The symptoms may be subjective or objective, or both. The recognition of the former comes from the history of the disease and the complaints of the patient. The latter, or the objective symptoms, are determined by the physical examination of the patient and the examination of the blood.

Anemia.—Anemia is characterized by a reduction in the number of red blood cells, or of their hemoglobin. In general we say an anemia is (A) primary, essential, or idiopathic, due to the absence of a well-defined cause, or (B) secondary, due to some recognizable blood destroying condition. The anemias may be suitably subdivided as follows:

$$\text{Anemia} \left\{ \begin{array}{l} \text{Primary} \left\{ \begin{array}{l} \text{Chlorosis.} \\ \text{Pernicious anemia.} \end{array} \right. \\ \text{Secondary} \end{array} \right.$$

The most causal observation may be sufficient for the recognition of anemia. The color of the skin, the appearance of the mucous membranes, and the evident breathlessness of the patient are gross indications of changes in the blood. On injury it is found that the patient is easily

prostrated, that there is breathlessness on exertion, that there is palpitation and perhaps cardiac oppression. The patient complains of neuralgias in various parts of the body. Headache is a more or less constant symptom, with the peculiarity that it increases when the patient ascends stairs, and is often throbbing or pulsating. The anemic subject has usually a poor appetite and suffers from gastralgia, although it may be remembered that the gastric symptoms of anemia are as often primary as secondary. Many of the symptoms which attend neurasthenia occur in the course of anemia.

On physical examination of the patient the appearances above indicated are observed, although grave anemias may be present, and yet the lips be bright red, the color under the nails fair, and the cheeks flushed. (See Color or Hue of the Surface.)

A study of the heart and bloodvessels usually yields the physical signs that attend anemia. Here again it must be remembered that considerable anemia may be present without the customary murmurs in the bloodvessels.

The Blood.—The final diagnosis of anemia rests upon an examination of the blood. The various forms of anemia give rise to blood changes in a measure peculiar to the respective variety. The primary anemias have characteristics which will be described in the special sections. The secondary anemias show the following blood changes.

In *acute secondary anemia* from hemorrhage the red corpuscles may be reduced to 1,500,000 while the hemoglobin is reduced to a degree greater than the red cells. The leukocytes are increased in number, the polynuclear forms being relatively much more numerous than the other varieties.

The red corpuscles are paler than normal. There is anisocytosis and some poikilocytosis. An excess of nucleated red corpuscles, or blasts, is seen in severe anemias. If the normoblasts are in excess, active regeneration is in progress; if the megaloblasts, there is revision to embryonal regeneration, a serious import in a case of anemia. In fatal anemia, the red cells are like those in the form just described, although nucleated red corpuscles are absent. The white cells are sometimes reduced in number; the mononuclear forms may be relatively numerous.

In *chronic secondary anemia*, the occurrence of polychromatophilia, anisocytosis, and poikilocytosis is constant and marked. Nucleated red corpuscles are not common, although a few of some kind are seen in the severe forms; usually large nucleated cells with karyokinetic figures occur. These corpuscles have pale-staining nuclei. The blood-platelet count is increased. There is usually moderate leukocytosis in secondary anemias, with increase in the polymorphonuclear elements.

For clinical purposes it is necessary to make a number of etiological divisions of chronic secondary anemia.

I. Toxic Anemias.—The poison may be developed in the body or it may be introduced from without. Toxemia is a factor in the anemias

Pernicious Anemia.—Pernicious, essential, Addison's, or Biermer's anemia is a severe anemia occurring without adequate known cause, running a progressive course with pronounced remissions and usually terminating in death. The *etiology* of the disease has not been determined satisfactorily. It occurs most frequently after the twentieth year and between that year and the age of fifty. Excluding the influence of pregnancy and parturition, it is twice as frequent in men as in women. Previous exhausting disease, chronic gastric and intestinal catarrh, great physical overexertion, exposure, great shock or fright, precede in certain cases the development of the disease. The disease is variously attributed to an infection, either bacterial or protozoal, to alimentary sepsis leading to hemolysis, or to a primary myelogenous disorder. It is probably due to faulty hematogenesis or hemolysis, or a combination of both.

SYMPTOMS.—The disease usually develops slowly and insidiously, the patient presenting the ordinary symptoms of anemia—pallor, weakness, shortness of breath, palpitation, venous murmurs, loss of appetite, and impaired digestion. As the disease progresses, the skin becomes a pale lemon hue, weakness and dyspnea increase; the patient has attacks of dizziness, faintness, and ringing in the ears; there may be slight edema; hemorrhages occasionally occur from the nose, the bowels, and into the retina. The hemorrhages are small and distinct in the skin and mucous membranes. There is no emaciation; the patient appears well-nourished. The long bones are often tender upon pressure. Secondary sclerotic changes in the spinal cord cause late symptoms of locomotor ataxia or lateral sclerosis. The urine is of low specific gravity, and usually contains an increased amount of urobilin. Diarrhea may be present.

Fever.—A peculiarity of the disease is the occurrence of fever of an irregular type. The temperature rarely rises higher than 102° or 103° F. in the evenings, and is followed by a morning remission. It is not usually present in the early stages of the disease, may be absent for weeks at a time when the disease is fully developed, and may cease entirely in the later stages.

Blood.—The blood changes in idiopathic anemia are characteristic, and are essential to the diagnosis of the disease. In brief they are: (1) very large reduction in the number of red-blood cells; (2) an absolute diminution in the amount of hemoglobin, but as compared with the number of red cells sometimes a proportionate increase; (3) considerable variation in the size of the cells, the average size of the cells probably being larger; (4) poikilocytosis; (5) nucleated red-blood cells; (6) degenerative cells.

Reduction in the number of red cells (oligocythemia) reaches a more extreme degree in pernicious anemia than in any other disease; the number often falls below 1,000,000. Poikilocytosis is present, but even more characteristic is an anisocytosis. The average size of the red cells seems to be increased, as is the proportionate amount of

hemoglobin in each cell, but microcytes are fairly common. Erythroblasts are almost always found, the megaloblast cells predominating. The so-called *blood crises* are characterized by a sudden transient increase in the number of circulating erythroblasts, usually megaloblastic in type and some slight increase in the number of ordinary erythrocytes and in the leukocytes. Polychromatophilia and granular degeneration of the red cells is also common. The leukocytes are usually diminished in number, though there is a relative lymphocytosis. The blood platelets are usually diminished.

DIAGNOSIS.—The most important diagnostic features of the disease are extreme oligocythemia, relatively high percentage of hemoglobin (color index high), a noticeable number of large nucleated red blood cells (megaloblasts), an average increase in the size of the cells, and all this without emaciation or discoverable local disease that might bear a causative relation to the anemia. Extremely characteristic are the periods of remission in which the blood picture approaches that of the normal, only to be followed by a relapse. The remissions gradually become less marked, shorter, and without much improvement in the blood pictures until death takes place. In addition, retinal, subcutaneous, and submucous hemorrhages, a urine with urobilin in excess, and vague gastric symptoms in the absence of organic disease, point to pernicious or idiopathic anemia. The distinction from severe secondary anemias cannot, as a rule, be based on the blood examination alone. The history and the physical signs must play a prominent part, while all possible causes of secondary anemia must be excluded. Carcinoma of the stomach or liver, cirrhosis of the liver, heart disease, renal disease, tuberculosis, parasitic anemias, can all be excluded by an ordinary careful study of the case.

APLASTIC ANEMIA.—This is a severe form of Addisonian anemia in which the bone-marrow shows aplastic (failure of formation of new cells) instead of hyperplastic or metaplastic changes. It differs clinically from the ordinary form by especially affecting young women; by its steady progression without remissions, causing death in a few months; by its tendency to cause subcutaneous or submucous hemorrhage and in its blood-picture, viz., a low color index, absence of erythroblasts, much poikilocytosis and anisocytosis, and a leukopenia in which the lymphocytes are much increased.

Leukemia (Leucocythemia).—A disease of the blood-making organs characterized by great and persistent increase in the white blood-corpuscles, by diminution in the number of red-blood cells, which are altered in shape and size, and display nucleated and degenerated forms, by a lessened amount of hemoglobin, and by changes in the spleen, lymphatic glands, and medulla of the bones. As a rule, the blood in leukemia presents one of two distinct types: (1) the *myeloid*, associated with great enlargement of the spleen, marked marrow changes, and little or no enlargement of other lymphatic tissue; usually a very chronic type; (2) the *lymphoid*, in which some one set of lymphatic

glands is enlarged, and the enlargement of the spleen sometimes equals that of the myeloid form. These cases may be acute or chronic.

The cause of the disease is unknown, some believe it is infectious in nature, others that it is neoplastic. Neither theory has been proved. The disease occurs twice as frequently in men as in women, and two-thirds of the cases appear between the twentieth and fiftieth years.

Myeloid Leukemia.—In the *myeloid* or so-called *splénomédullary* form of the disease the onset is insidious. The patient may first notice gradual weakness and loss of strength. Occasionally profuse hemorrhage from trifling causes, as the drawing of a tooth, may be the earliest symptom. Oral symptoms resembling any acute mouth or throat infection, may be the first manifestation of the disease. More frequently, however, the patient first notices that the spleen steadily enlarges, though it may attain considerable size before the patient becomes aware of it. The enlargement is not usually painful, but gives rise to a feeling of distention, weight, and dragging. There may be tenderness on palpation and pressure, and sometimes the patient complains of sharp, stabbing pains, due either to attacks of local peritonitis or to sudden enlargement of the spleen and consequent stretching of the capsule. The splenic enlargement is uniform, so that its shape and characteristic notch are unchanged. Not infrequently the enlargement is so great as to fill the left hypochondriac and iliac regions, and reach beyond the middle line toward the right groin. Sometimes a venous hum can be heard over it. As the result of this enlargement the diaphragm is pushed upward, increasing the dyspnea already caused by anemia, and sometimes inducing palpitation. The gastric functions are disturbed from pressure; vomiting and other symptoms of dyspepsia are common.

A rise in temperature is a very common symptom. The fever is of irregular type, usually with nocturnal exacerbations, the temperature not often rising above 102° F. The febrile type may be intermittent or remittent, and sometimes there are periods of apyrexia. The apyrexia is said to be most marked toward the close of the disease. Such cases run a usually rapid course, marked by considerable dropsy and extensive hemorrhage.

As the disease progresses weakness increases; anemia becomes more intense; edema, ascites, or hydrothorax occurs; hemorrhage from the nose, gums, bowels, stomach, lungs, or uterus further exhaust the patient; digestion is poor and diarrhea is common. Headache and tinnitus are frequent symptoms, occasionally delirium and coma may occur, and deafness is not uncommon toward the close of the disease. The eyes may be the seat of leukemic retinitis.

The liver is enlarged, often to a considerable degree, but without special symptoms. The same is at times true of the lymphatic glands and other adenoid tissues. The long bones may be tender upon pressure.

The Blood.—The most characteristic and important changes, from a diagnostic point of view, occur in the blood. There is always, sooner

or later, a secondary anemia which is characterized by a notable number of normoblasts. The platelets are increased as is also the coagulation time. The number of white cells of all varieties is greatly increased, but a large percentage of these increased cells is made up of mononuclear, finely granular cells, the *myelocytes*, or marrow cells. These cells are not ameboid, a distinctly noticeable feature. The granules are fine and neutrophilic; the nucleus is oval. As many as 1,000,000 white cells (average 450,000) may be counted in a cubic millimeter of blood, the myelocytes constituting 30 to 50 per cent. of the total number. Instead of there being one white cell to 300 or 500 red, the ratio falls as low as 1 to 5 or 1 to 3, or even lower. Even in periods of comparative well-being, when the total leukocyte count may be nearly or quite normal, a high percentage of myelocytes still remains and distinguishes leukemia from other conditions in which the white cells are increased. In ordinary leukocytosis myelocytes are absent and an increased percentage of the polymorphonuclear leukocytes is regularly seen.

Not only do the white cells greatly increase in number, but they vary considerably in size and react differently to staining fluids.

The variations in the leukocytes in this disease are as follows: (1) the small mononuclear, especially, and also the polymorphonuclear elements are relatively diminished, although absolutely increased; (2) the great difference in the size of the multinuclear elements; (3) the presence of the myelocytes; (4) the presence of a normal or but slight, relative increase of eosinophiles in so extensive an increase of leukocytes and the presence of eosinophiles with a single round or oval nucleus, the eosinophilic myelocytes, rarely seen in any other disease; (5) large mononuclear elements with karyokinetic figures (Müller); (6) mast cells increased relatively and absolutely.

The essential points in the diagnosis of myeloid leukocythemia are (1) a marked excess of leukocytes in the blood, with a high percentage of myelocytes, the ratio of white to red falling below 1 to 50 or 1 to 20, the individual leukocytes varying in size and characteristics, as already described; (2) enlargement of the spleen; (3) the occurrence of hemorrhages and dropsies unexplainable by disease of the heart, kidneys, or other organs; (4) the symptoms of a severe grade of anemia, as dyspnea; (5) leukemic retinitis; (6) anemic fever; (7) the presence of eosinophilic myelocytes, mast cells, and nucleated red-blood cells.

Lymphoid Leukemia.—The lymphocytic form of the disease is not so common. It may be chronic or acute. The former is characterized by enlargement of groups of the lymphatic glands and by the same general symptoms and signs as the myeloid form (excepting the great increase in the size of the spleen and the liver). The great increase in leukocytes is in the proportion of the lymphocytes, which are of the small variety and average about 90 per cent. of the leukocytes present, though large lymphocytes are also found. The granulocytes are abso-

lutely as well as relatively decreased. The total increase in the colorless elements is not so excessive; 300,000 per cm. is an extreme. Eosinophiles and nucleated red cells are rare. The myelocyte is not usually present. Acute leukemia of the lymphoid type frequently show the greatest increase in the large mononuclear elements (lymphocytes); the main features are high fever, often a pronounced angina, marked tendency to hemorrhage, a duration of symptoms of only a few weeks, late swelling of the glands, and a rapidly progressing anemia, occasionally with nucleated red cells.

Atypical Leukemia.—1. *Anemia infantum pseudoleukemica* of v. Jaksch is disputed as a clinical entity; an intense anemia, leukocytosis, large spleen and liver, a blood condition with certain characteristics of both leukemia and pernicious anemia or even of secondary anemia are to be seen. It is remembered that the infantile hemopoietic system responds readily to any influence with a result far more striking and varied than in the adult. Many of these cases may be leukemias, pernicious anemias, or anemias secondary to rickets or syphilis.

2. *Chloroma* is an atypical form of lymphocytic leukemia in which there is myeloblastic hyperplasia. The resulting tumors have a greenish tint and give rise to swelling over the affected bones or the tissues, as well as to certain pressure symptoms (exophthalmos, headache, bone pains). The blood changes and the symptoms are essentially like those of acute lymphoid leukemia.

3. *Leukanemia* is a term applied to a group of symptoms which are associated with the blood-picture of some of the features of leukemia and some of pernicious anemia. The term is redundant and probably only describe atypical forms of leukemia associated with profound anemia.

Erythremia.—*Polycythemia with Cyanosis.*—Vaquez's disease or Osler's disease is a disease of the hemopoietic tissues, characterized by persistent cyanosis, enlarged spleen, and polycythemia. The visible mucous membranes and the skin of the face and hands have a rather peculiar purplish mottled appearance which may in long-standing cases become pigmented. The patient may notice headache, tinnitus, hebetude, progressive and gradual weakness, and abdominal fulness. Hemorrhage from the mucous membranes occasionally occurs. The spleen, and at times the liver, is enlarged. The blood-pressure is elevated and the blood shows a marked erythrocytosis (8,000,000 to 13,000,000), increase in hemoglobin (120 per cent. to 200 per cent.), and a slightly increased or normal leukocyte count with a relative increase of the polynuclear neutrophils.

HEMORRHAGIC DISEASES

Purpura.—A term applied to the small spontaneous hemorrhages which occur in the skin or mucous membranes. Its pathogenesis is

unknown, though commonly it is symptomatic of some obvious disorder. The so-called primary or idiopathic purpura has been divided into the simple, hemorrhagic, rheumatic, and abdominal types. All these types are probably only degrees of severity or various manifestations of the same disease.

Idiopathic Purpura.—In *simple purpura* (*purpura simplex*) the hemorrhages are limited to the skin. They consist of (1) bright red spots, varying in size from that of a pin-head to that of a ten-cent piece; these spots are under the skin and are unaffected by pressure; they fade gradually from red to yellow and disappear; (2) larger spots or streaks called vibices; (3) ecchymoses. The disease is said to be most common about the age of puberty. It may come on in the midst of apparent health, or it may follow an illness, as typhoid fever. Purpura occurs especially upon the legs, the standing position seeming to favor its development. The hemorrhages appear in successive crops.

In the *hemorrhagic form* (*morbus maculosus*), hemorrhage occurs from the mucous membranes as well as into the skin. The onset of these cases is sudden, although there may be a day or two of depression, lassitude, headache, and nausea. The first symptom noticed is generally fever, which is apt to be moderate, then an eruption is detected, and for a day or two the patient may seem to be only slightly ailing. Copious epistaxis may now occur, or hematemesis, hematuria, hemoptysis, or bleeding from the bowel, or all of these and other hemorrhages may occur the same day. The temperature may be only moderately raised, or it may reach 104° to 105° F., or even a higher point. The pulse at first is frequent (120 to 140), but of good volume and tension. Subsequently, in unfavorable cases, it becomes thready and very frequent. Hemorrhage may also occur into the choroid and brain substance, with blindness and paralysis as sequels. It may also occur into the uvula or tonsil.

The degree of anemia depends upon the copiousness of the hemorrhage and the length of time the disease lasts. Sometimes the hemorrhages cause great exhaustion, with a tendency to collapse. In this as in all forms of purpura the clotting time of the blood is greatly delayed.

Rheumatic Purpura (*Peliosis Rheumatica*—*Schönlein's Disease*).—The peculiar features of this type are tender and swollen joints, edema of the subcutaneous cellular tissue, and purpura, associated frequently with urticarial wheals and intense itching (*purpura urticans*). The subcutaneous hemorrhages consist of petechia, vibices, and ecchymoses. There may be such large hemorrhages into the penis, scrotum, and uvula as to result in gangrene and slow separation of the dead tissue by ulceration. Epistaxis may occur, but copious hemorrhages from the stomach, the bowel, or into the kidney or other organs are rare. The duration is apt to be long, convalescence being delayed by repeated outbreaks of purpura, with multiple arthritic symptoms and edema.

Osler has called attention to the frequent association of purpura,

angioneurotic edema, urticaria and erythema occurring chiefly in children, and with a pronounced tendency to frequent relapses and recurrences.

Abdominal Purpura (Henoch's Purpura).—This type resembles hemorrhagic purpura, but is further characterized by gastro-intestinal crises of severe pain, vomiting, diarrhea and hemorrhage, enlargement of the spleen, with occasionally arthritis and in many cases, acute nephritis.

Diagnosis.—Primary purpura is distinguished from scurvy by the absence of antecedent debility and anemia, of spongy gums, and of brawny induration in the limbs. As a rule, the cutaneous hemorrhages are larger in scurvy than in purpura.

Purpura is distinguished from acute infectious diseases by the absence of severe constitutional symptoms which characterize the graver forms of these diseases—in which alone a purpuric eruption is likely to be severe enough to cause doubt.

Hemophilia is distinguished by the history the patient gives of being a bleeder by heredity.

Secondary or Symptomatic Purpura.—Purpura is frequently a symptom of the following conditions and states, the hemorrhages usually occurring as petechia or ecchymoses.

1. *Infections*, notably acute endocarditis, typhus, cerebrospinal fever and sepsis; less frequently measles, smallpox, scarlatina, syphilis, etc.

2. *Intoxications.*—(a) Exogenous toxins as snake bite, drugs (iodides, bromides, quinine, belladonna, ergot, and copaiba); (b) endogenous toxins as those formed in the course of jaundice and nephritis.

3. *Cachectic States.*—Senility, cancer, sarcoma, tuberculosis, Hodgkin's disease, leukemia, pernicious anemia, and starvation.

4. *Neurotic Conditions* (myelopathic purpura).—Myelitis, tabes, neuralgia, and hysteria (stigmata).

5. *Mechanical Conditions.*—Prolonged venous stasis, as in whooping cough paroxysms, epileptic seizures, heart disease, or after the application of tight bandages.

Hemophilia.—When hemorrhage recurs and is not readily checked; when it occurs spontaneously or follows a slight trauma; when it occurs both in skin and mucous membranes; when there is a family history of hemorrhage and it occurs in males it is likely to be due to hemophilia. When to these symptoms are added swellings of one or more of the large joints, occurring with or without pain (hemarthrosis), sometimes with heat, redness, and fever resembling tuberculosis, or at times in a form resembling arthritis deformans, the symptom complex is complete.

The frequent seat of hemorrhage is the nose, and next the gastro-intestinal tract and kidney. The bleeding is from the capillaries; it may prove fatal in a few hours, or last for days or weeks, with final recovery. Intense anemia follows the prolonged hemorrhage, but the blood is replaced with remarkable rapidity. All operations, even the most trivial, are extremely dangerous in bleeders. Circumcision,

extraction of teeth, and leeching are credited with the most deaths by Grandidier. The disease is transmitted through the female members of a family to the males. It is in all probability due to the absence of one of the elements necessary to produce clotting of the blood with consequent insufficient fibrin formation.

Diagnosis.—This is readily made upon the trend of symptoms of *inherited tendency to bleed* in males. The coagulation time of the blood is slower than in any other disorder. It is much delayed or the blood may completely fail to clot.

Hemorrhage in the Newborn.—Hemorrhages in the newborn occasionally occur as a result of birth injuries, septicemia acquired during birth, or congenital syphilis. The hemorrhage may come from the umbilicus, the nose, the gastro-intestinal tract, or the genito-urinary tract.

Hemorrhage in the newborn may be a manifestation of one of the following three specific diseases:

1. *Epidemic Hemoglobinuria* (Winckel's Disease).—This disease is probably infectious in origin. It is characterized clinically by fever, jaundice, and cyanosis, suddenly appearing within the fourth to tenth day of life, followed by dyspnea, vomiting, diarrhea, hematuria, and methemoglobinuria and small hemorrhages into the viscera and tissues. The disease occurs in institutions and runs a rapid course, usually terminating fatally.

2. *Acute Fatty Degeneration in the Newborn* (Buhl's Disease).—Although very similar to Winckel's disease, there is a greater tendency to external hemorrhage and inanition in this lethal affection, which shows, postmortem, marked fatty degeneration of the internal viscera.

3. *Morbus Maculosus Neonatorum.*—The "hemorrhagic disease of the newborn" clinically resembles closely the hemorrhagic form of purpura (*morbus maculosus*), but there is a greater tendency to hemorrhage from the bowel. The disease appears to be self-limited and is frequently associated with fever.

DISEASES OF THE DUCTLESS GLANDS

The Pituitary Body.—Diseases of the pituitary body are recognized either by symptoms suggestive of a brain tumor (*q. v.*) or by symptoms due to alteration in the functions of the gland or by a combination of both. The anterior lobe is supposed to elaborate an internal secretion, a hormone which in some way affects bodily growth and sexual development. The functions of the posterior lobe in some way are related to metabolic processes, more especially the assimilation of sugar.

Hypersecretion of the anterior lobe of the gland in the adult causes acromegaly (see page 146), and when congenital, gigantism. Intolerance for sugar, loss of flesh, elevation of the blood-pressure, and a moist skin are characteristic of hypersecretion of the posterior lobe.

Congenital hyposecretion of the anterior lobe causes infantilism,

which is characterized by imperfect growth and incomplete development of the sexual organs, while hyposecretion in later life results in loss of sexual characteristics. Hyposecretion of the posterior lobe leads to a marked increase in the tolerance of sugar, with a tendency to adiposity, psychic disturbances and somnolence, excessive thirst and polyuria, subnormal temperature, slow pulse, and lowered systolic blood-pressure. Alterations in the functions of the lobes are usually associated, but not necessarily is there a similar alteration in function. Frequently there is a hypersecretion of one lobe and a hyposecretion of the other, *e. g.*, acromegaly is frequently associated with symptoms of hypersecretion of the anterior lobe and hyposecretion of the posterior lobe.

The Thymus.—The functions of this gland are unknown, though it is supposed in some way to be connected with the organs of internal secretion. The only condition of the gland of clinical interest is enlargement or hypertrophy. This is usually associated with status lymphaticus. It may be the cause in children of laryngismus stridulus or of distinct asthmatic attacks (thymic asthma). Sudden death from asphyxia (*thymustod*) in children without previous respiratory difficulty and without adequate cause (anesthesia, etc.) is frequently found to be associated with an enlarged thymus and status lymphaticus. An enlarged thymus is probably present if there is general lymphatic enlargement associated with dulness at the upper part of the sternum and slightly to the right and left, extending to the area of cardiac dulness. A palpable mass may sometimes be felt above the sternum, when the head is retracted. The x-ray will positively disclose the presence of an enlarged gland.

The Thyroid Gland.—The thyroid gland may be enlarged from simple goitre or from exophthalmic goitre. Enlargement may also be due to inflammation, abscess and morbid growths.

Exophthalmic Goitre (*Graves', Parry's, or Basedow's Disease*) is a condition notably largely due to hyperthyroidism or hypersecretion of the thyroid gland (largely iodothyron), characterized by *tachycardia*, *tremor*, *exophthalmos*, with associated ocular symptoms, and *enlargement of the thyroid*. The condition is supposed by some to be primarily in the nervous (sympathetic) system; by others to be simply a disease of the thyroid gland alone. Graves' disease begins slowly in most cases, though occasionally the onset is acute. The disease is commonly subject to remissions and exacerbations and runs a very chronic course. It is far more common in women than in men. It may develop at any age, but is most common in early adult life. A neurotic heredity, exhausting disease, infections, general debility, and anemia are predisposing causes, while sudden fright or shock is said to be a common exciting cause.

Tachycardia.—Attacks of palpitation may recur at intervals. The frequency may not be over 100 or 120 in the early stages. Later, however, the pulse beats 160 to 180 or even 200 per minute. It is small and regular. In time the heart becomes hypertrophied and dilated and there is often a loud basic, systolic murmur; the larger arteries an

even sometimes the smaller ones show the vascular distribution by increased pulsation, sometimes with thrill.

The Thyroid Gland.—The thyroid enlargement may be very slight or extreme. The size of the goitre seems to have no relation to the severity of the symptoms. The right lobe may be larger than the left. The enlargement is painless, soft, and compressible; it may pulsate with or without thrill, and over it can be heard vascular murmurs.

Exophthalmos.—Prominence of the eyes is the most conspicuous feature of well-marked cases. Like enlargement of the thyroid, it varies in degree, but rarely is wholly absent. The protrusion allows the white sclerotic to show above and below the cornea, giving the eyes an unnatural, startled, staring appearance (Dalrymple's sign). When the eyeball is simply directed downward the upper eyelids do not follow, but remain spasmodically elevated or lag behind the movement of the eyeball (von Graefe's sign). The eyeball may become inflamed and even slough from inability to close the eyelids. In rare cases one eyeball alone is affected. Infrequent and incomplete reflex winking (Stelwag's sign), inability to evert the upper lid (Gifford's sign), tremor of the upper lid when it is closed gently (Rosenbach's sign), insufficiency of the internal recti muscles (Moebius' sign), and numerous other ocular signs are present.

Tremor.—Fine involuntary muscular tremors, eight to nine a second, are present in nearly all the cases. They are usually symmetrical but vary slightly in degree in the different extremities. The mentality is one of irresponsibility, unconcern as to the gravity of the disease, and want of concentration.

In addition to these characteristic symptoms, loss of flesh and strength, periods of pyrexia of irregular type, impaired appetite, diarrhea, and dependancy are observed. The diarrhea is characterized by the passage of three or four large, fatty stools a day. The menstruation is apt to be disturbed. Tinnitus aurium, headache, and vertigo are not uncommon, and sometimes there is profuse sweating. A restless, nervous excitement (Charcot) is very common, edema of the feet is often seen. Transitory angioneurotic edema of the eyelids, the face, hands, and the supraclavicular and infraclavicular regions occurs. Transient erythemas, excessive sweating, flushing, urticaria, and other vasomotor phenomena are common. A more or less pronounced

FIG. 180



Exophthalmic goitre. (Original.)

secondary anemia is present, associated with a relative and at times absolute lymphocytosis. Polyuria is frequent and there is a tendency to transitory glycosuria.

Diagnosis.—Well-developed cases are readily recognized. The early cases by hyperthyroidism without exophthalmos and with questionable enlargements of the thyroid present difficulties in diagnosis, but the association of tachycardia, tremor, nervous symptoms, and vasomotor phenomenon are most suggestive.

Myxedema.—Myxedema is a condition caused by a total or partial absence of the thyroid secretion (hypothyroidism). The following varieties are seen: (1) spontaneous myxedema of the adult; (2) postoperative myxedema (cachexia strumipriva); (3) infantile myxedema (cretinism).

1. *Spontaneous myxedema* is a disease of mature life and occurs most frequently in women. It is characterized by dense unelastic swellings which are general and which do not pit on pressure. The arms are more frequently affected than the fingers; the legs more than the feet. Usually the swellings are irregular. In some cases supraclavicular paddings may be the most marked. There is a striking change in the appearance of the face, particularly the nose and forehead. The nose becomes thickened, the forehead more prominent and overhanging. The outline of the face is rounded, and the term "full-moon" is applied to it. The skin is thickened, dry, and rough, somewhat translucent, pale or yellow in color, and of a doughy consistence, but with a moderate degree of elasticity. The hands change in shape, become square or spade-shaped, and the fingers clubbed. The nails become brittle and disturbed, the hair dry, harsh, and may fall out. The body weight becomes much increased; marked nervous and mental symptoms arise. Speech is thick and hesitating, the memory feeble. The intellect is dull and irresponsive; memory is weakened; the temper irritable. Sensibility is impaired, particularly the pain sense. The perspiration is diminished. Abnormal sensations of heat and chilliness are complained of, as well as other paresthesias. The patient is anemic, the temperature is subnormal, the heart's action is weak, the respiration sluggish. Breathlessness on slight exertion is pronounced, and exertion itself is very difficult; while there is a greater sense of fatigue than the exertion and the condition of the organs would warrant. The muscularity is enfeebled. There is impairment of appetite, indigestion, and flatulence. Albuminuria and glycosuria are common.

2. *Postoperative Myxedema.*—This occurs after the removal of a part or the whole of the thyroid gland. The symptoms are analogous to the adult form of myxedema.

3. *Infantile Myxedema.*—Cretinism may be congenital or acquired, occurring sporadically or endemically. The arrested physical and mental development are usually not noted until the end of the first or during the second year of life. The head is small, the fontanelles open, the face round, bloated, the lips are thick and the lips are

thick and swollen; the tongue is enlarged and protuberant; the expression idiotic; the body is dwarfed; the legs are short; the abdomen is large. The skin is dry and rough and myxedematous. The hair is brittle. The temperature is subnormal and the pulse slow. Cretins are usually idiotic to a greater rather than a less extent.

As the case advances mental and physical failure becomes more pronounced, the patient becomes subject to hallucinations, and extremely irritable.

The Parathyroid Glands.—(See Tetany.)

The Adrenals.—Addison's disease is a condition probably due to insufficiency of the adrenals as well as the rest of the chromaffin system generally, the result of destructive lesions, usually tuberculous. The specific secretion of this system, adrenalin, has for its chief function the maintenance of blood-pressure and of muscular tone. Disease of this system, therefore, causes gradual loss of strength without much loss of weight, and cardiovascular asthenia. The disease is further characterized by pigmentation of the skin and gastro-intestinal symptoms. It occurs most frequently during the active period of life, from the age of twenty to forty years, and nearly twice as often in males as in females. The disease begins insidiously with gradual and progressive loss of strength. It becomes evident from the patient's languor, weariness on slight exertion, and inaptitude for mental effort that he is suffering from some exhausting disease. The most characteristic symptom is the extreme prostration without obvious cause. The appetite is impaired or there is more or less discomfort at the epigastrium and occasional vomiting, often severe and frequently attended by headache. Dull pains in the head, back, and abdomen are not uncommon; neuralgic pains in the limbs may be noted, and some tenderness on pressure in the lumbar region.

The pulse is extremely small and feeble; in the later stages it may be absent at the wrist. The systolic blood-pressure is rarely above 100 mm. of Hg. The discoloration of the skin appears gradually and is the most striking symptom of the disease when it is well-marked. The external surfaces are changed in hue, and delicate portions of the skin underneath the clothing are also bronzed. The discoloration is not removed by pressure. The areas are irregular in shape. The skin is soft and pliable. The pigmentation is never seen in the cornea or in the nails. Bronzed areas in sharply circumscribed patches are also seen in the visible mucous membranes. Sometimes the whole body becomes a walnut-juice color, a bronzing which is deeper in exposed surfaces. At times only portions of the body are discolored, in which case the dark hue shades off gradually into the normal hue of the skin. At times the bronzing and other characteristic symptoms of Addison's disease are associated with tuberculosis in other organs. Conversely, in cases of phthisis in which there is bronzing, tuberculous disease of the adrenals may be suspected.

The Lymphatic Glands.—Most disorders of the lymphatic glands are considered in Chapter XIV. (Examination of the Lymph Glands.)

Hodgkin's Disease (pseudoleukemia) is a disease of unknown cause (possibly an infection), characterized by progressive enlargement of the lymph nodes, and by the gradual development of a severe anemia with cachexia, and terminating fatally. It is a disease especially common in young males.

Symptoms.—The first symptom noted is *enlargement of the glands* of the neck, but sometimes the inguinal, less frequently the axillary glands, are first enlarged. The enlargement is painless and progressive, appearing first on one side of the neck and extending under the jaw to the opposite side. The tumors at first are distinct, and movable under the skin; eventually they lose their separate identity, and coalesce into large masses. Other glands in remote parts, as the axilla and groin, retroperitoneum and arm, are affected.

It is difficult to determine accurately the beginning of the disease; sometimes a long period of latency follows the early glandular swelling; sometimes a general anemia precedes any noticeable swelling of the glands; sometimes the disease runs an acute course, ending fatally in two or three months; again, the onset of the disease may be marked by fever and constitutional symptoms, and the glandular enlargement appears later.

The spleen becomes greatly enlarged, but rarely attains the dimensions common in leukemia. Other adenoid tissue in the intestine, tonsils, posterior nares, and even the thymus, may enlarge and give rise to pressure symptoms.

Fever is a very constant symptom, but the type is not constant. It may be intermittent, remittent, relapsing, or continuous. A curious form of paroxysmal relapsing fever has been described by Ebstein. In the interval, which may last ten days or more, the temperature is normal. The paroxysms last from ten to fourteen days.

A severe anemia of the ordinary secondary type develops progressively. It may be rapid in onset or appear gradually but ultimately leads to a pronounced cachexia. The leukocytes may be slightly increased and the differential count is unchanged though there is often a tendency to a relative lymphocytosis.

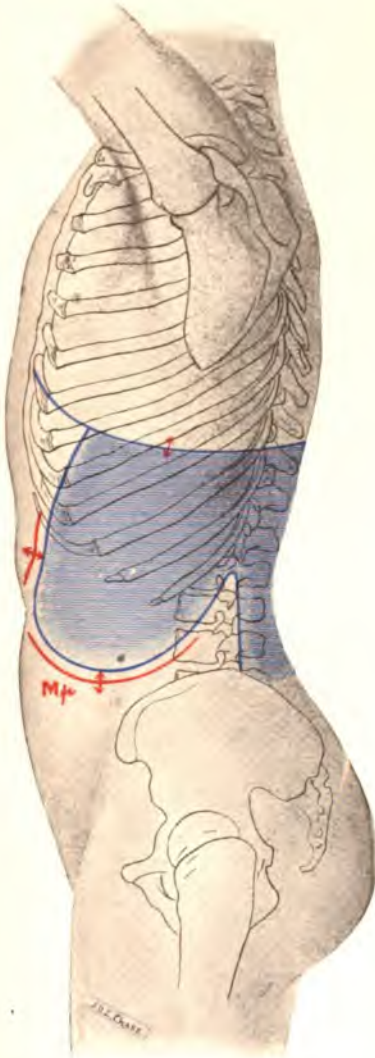
There are local symptoms due to pressure—cerebral anemia from pressure on the carotids—cerebral congestion from pressure on the veins of the neck; disturbance of the heart from pressure on the pneumogastric; deafness; difficulty in deglutition and mastication; and pleural, peritoneal, and pericardial effusions.

The *duration* of the disease is from six to eighteen months, death usually resulting from exhaustion.

The diagnosis is based upon the occurrence of painless, discrete progressive enlargements of the lymph nodes associated with a normal leukocytic picture.

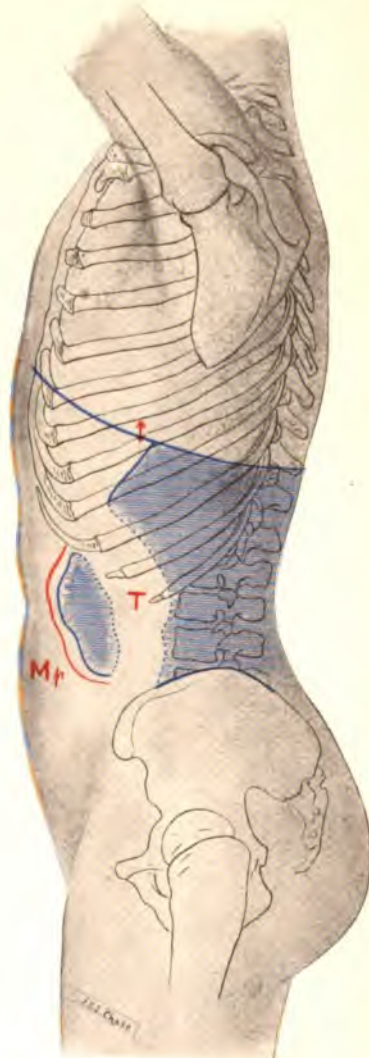
PLATE X

FIG. 1



Enlargement of the Spleen.

FIG. 2



Tumor of the Left Kidney.



Tuberculous or *syphilitic adenitis* may resemble the condition, and in the absence of other demonstrable manifestations of these conditions, resource must be had to the specific tests. *Lymphosarcoma* is usually a painful, rapidly developing swelling, but may so mimic Hodgkin's disease that a portion of the gland may have to be excised and microscopically studied to make a diagnosis. The same may be said of *leukemia* during the periods that the blood is normal.

THE SPLEEN

Disease of the spleen is rarely primary, but occurs as a rule secondary to disease elsewhere in the body. The symptoms are those of the original condition, the secondary involvement of the spleen being recognized only by the enlargement of the organ. Enlargement of the spleen may be acute or chronic.

Acute Enlargement (*Acute Splenic Tumor, Acute Splenitis*).—This occurs in certain infectious diseases, particularly typhoid fever, typhus fever, smallpox, septicemia, and erysipelas. It occurs also in diseases caused by animal parasites, as malaria, secondary syphilis, kala-azar, relapsing fever, and trypanosomiasis. A rare form of enlargement is the result of emboli lodged in the spleen. The spleen increases rapidly in size, with evidence of local pain and tenderness; fever is present, and if suppuration ensues, the fever becomes hectic.

Chronic Enlargement.—Chronic enlargement of the spleen arises as a result of various blood diseases, as leukemia, splenic anemia, infantile anemia, chronic polycythemia with cyanosis, pernicious anemia, and chlorosis. In leukemia it frequently attains an enormous size, extending well into the abdomen and at times as far down as the ileum. Portal obstruction, secondary to cirrhosis of the liver, pylephlebitis, etc., or mechanical damming-back of the venous blood by disease of the heart or lungs, may also cause chronic enlargement of the spleen, as do also amyloid disease and rickets. A splenic tumor due to the above causes is a symmetrical enlargement. If acute, it is soft and flabby; if chronic, firm and hard. Primary disease of the spleen is rare and causes characteristic irregular enlargements of the spleen, as in simple or dermoid cysts, primary tumors or abscess of the spleen. Irregular enlargements are also found in a few secondary conditions, *e. g.*, gummata of the spleen and hydatid cyst. The chronic enlargements are characteristically symptomless unless the enlargement is pronounced, when dragging and dull pain in the left side is experienced. In *perisplenitis* the spleen is enlarged, irregular, and often a distinct friction rub can be heard over it. Pain or discomfort limited to the region of the spleen is the chief symptom.

Diagnosis of Splenic Enlargement.—Enlargement of the spleen can be distinguished from enlargement of the left kidney by the greater movability of the spleen, by the area of dullness extending up to the

ninth rib or higher, and by the palpation of the sharp edge of the spleen. A kidney tumor is crossed by the colon and hence its dulness is obscured by the tympany of the bowel. A splenic enlargement is differentiated from an enlargement of the left lobe of the liver by the following procedure; after the anterior edge has been found, pressure with the other hand posteriorly will bring the spleen forward, which would not occur if the suspected enlargement was in the liver.

Primary Splenomegaly with Anemia (*Banti's Disease, Splenic Anemia*).—A primary disease of the spleen of unknown cause characterized by anemia, a tendency to hemorrhage and great chronicity. The condition is usually succeeded by a secondary cirrhosis of the liver, with ascites and jaundice.

Symptoms.—The enlargement of the spleen usually first calls attention to the condition. It is uniformly much increased in size, smooth and painless. The blood-picture will establish the diagnosis. There is a pronounced secondary anemia with a low color index and a leukopenia. Hemorrhages are common, the bleeding, as a rule, coming from esophageal varices. Late in the disease ascites develops, the result either of the splenomegaly or of secondary cirrhosis of the liver.

Diagnosis.—The diagnosis is, as a rule, readily made. With an enlarged spleen in pernicious anemia, the question of the differentiation of the two conditions is settled by the color index, low in Banti's disease—high in pernicious anemia. Other chronic enlargements of the spleen are differentiated by concomitant symptoms. Primary splenomegaly may at first glance be confounded with the Gaucher type of splenomegaly (primary endothelioma of the spleen) or with the splenomegaly of familial jaundice, but the confusion should not persist after the blood-count has been made.

Splenomegaly with Familial Jaundice.—In this condition there is persistent uniform enlargement of the spleen associated with persistent slight jaundice without clay-colored stools and with urobilinuria. The condition is strictly familial; it does not apparently affect the general health of the patient.

CHAPTER XXXVI

DISEASES OF THE NOSE AND LARYNX

DISEASES OF THE NOSE

Disturbances of the Sense of Smell.—Loss of smell, or anosmia, occurs to a moderate degree in all the inflammatory and obstructive diseases of the nose. The intensity depends upon the degree of change in the mucous membranes. Anosmia may also be due to disease of the olfactory nerve or its centre. Thus lesions of the uncinata may cause it, or destructive lesions of the olfactory bulb or tract as in fracture of the anterior fossa of the base.

Parosmia is the perception of abnormal odors, and may be due to irritative lesions in the olfactory tract or may be a neurosis or solely a psychical difficulty, as in hysteria.

Hyperosmia, abnormal olfactory sensitiveness, is frequently an associated symptom of neurasthenia and hysteria.

The subjective and objective symptoms of disease of the nose are due in general to inflammation, malformation, morbid growths, and foreign bodies. They are recognized by their subjective and objective signs, by rhinoscopic inspection, and by bacteriological and microscopic examinations. When secondary, both acute and chronic inflammations may be due to infection. To the acute secondary varieties belong the acute catarrh of measles, glanders, hay fever, or influenza; to the chronic belong syphilis and tuberculosis.

Simple Acute Rhinitis.—**Acute coryza**, or "cold in the head," is an infection caused by the *Micrococcus catarrhalis* alone or with other bacteria, starting in the mucous membrane of the nose and often involving all the upper air passages. It is ushered in with a feeling of lassitude, aching in the back and limbs, feverishness, and a sense of fulness in the nostrils, with sneezing. After twenty-four hours an irritating discharge begins. During this time the malaise has increased. The pain in the forehead and cheeks has become more pronounced, and a nasal twang is given to the voice. The fever continues, the temperature reaching 101° F. in the more pronounced case with thirst and loss of appetite. At the height of the fever, in twenty-four to forty-eight hours, a crop of herpes very often develops on the lips. The general symptoms then subside and the local symptoms change. The discharge becomes thick and purulent, the fulness continues, but the pain is diminished. The inflammation often extends to the tear ducts and to the eyelids. The latter are congested and smart exceedingly.

Very frequently, also, the inflammation extends to the pharynx, causing soreness of the throat and stiffness of the neck, and even the larynx may be involved. A slight deafness may result from the inflammation extending into the Eustachian tubes. *Syphilitic coryza* is seen in infants and young children affected with hereditary syphilis. The nostrils are swollen and red at the edges, sometimes completely occluded, causing oral respiration and inability to take the breast or bottle. Pustules, fissures, and ulcers are found in the nose and at the margin of the orifices. They are also seen in the pharynx and larynx. Hemorrhages may occur. Other evidences of hereditary syphilis are present.

Hay Fever.—Hay fever is an acute affection, probably in most cases a manifestation of a local anaphylactic reaction to certain pollens, ushered in by paroxysmal sneezing, itching, and smarting of the inner canthus of each eye, or of the throat or nose. After hours or days of sneezing, coryza develops. The disease continues for a varying length of time, and is more pronounced at certain seasons of the year, particularly the late fall, tending to recur at the same time each year. Coughing may be an additional symptom and paroxysms of asthma may develop which are hard to distinguish from true bronchial asthma. While the attack is usually excited by vegetable emanations, other emanations may also induce it. Certain conditions of the nasal mucous membrane predispose to the attack. Local inflammation of the nose or obstructive diseases from hypertrophies are primarily present. To the exciting cause and the local predisposing cause may perhaps be added a neurotic factor. *Asthma* may be due to disease of the nose, but the only proof that it is of nasal origin is that it disappears after the nose has been treated for the various ailments that are supposed to cause it.

Chronic Rhinitis.—Four varieties are distinguished, to all of which the term nasal catarrh is applied. In one there is hypertrophy of the turbinated bones; in the second there is extension of the disease to the postpharynx—chronic postnasal catarrh; in the third there is absolute dryness of the mucous membrane—rhinitis sicca, or dry catarrh; in the fourth there is atrophy of the mucous membrane—atrophic rhinitis or ozena.

Chronic Hypertrophic Rhinitis.—The affection comes on gradually after repeated acute attacks of coryza. The only symptoms may be slight fulness in the nose and a little hoarseness of the voice. In more advanced stages the symptoms of stenosis are marked, the patient snores and breathes through the mouth, and the voice becomes nasal. There is a constant discharge of mucus backward into the pharynx, causing hawking. The hearing is frequently impaired, as well as the taste and smell. The discharge often affects the larynx, causing an irritating cough. The hypertrophied tissue on the turbinated bones and the pressure of the bone on the septum may lead to reflex attacks of asthma.

Chronic Postnasal Catarrh.—Chronic postnasal catarrh is an extension of the rhinitis into the pharynx. It is distinguished by discomfort

or pain in the soft palate and posterior nares. There are tingling and a sense of fullness at the root of the nose, with frontal headache; the patient complains of a bad taste in the back of the mouth, and of constant flow of thick secretion into the pharynx, causing snoring and hawking. The same perversion of the senses of taste, smell, hearing, and of the voice occurs as in acute rhinitis. Headache seems to be due to the condition of the pharynx.

Dry Catarrh, or Rhinitis Sicca.—This is also chronic in its course, and is accompanied by tingling and dryness of the nostrils. A faint, musty odor is detected, but there is no discharge or sense of obstruction. In severe cases there may be sharp pain in the nose extending to the forehead.

Atrophic Rhinitis, or Ozena.—The disgusting, foul odor of the breath is characteristic, and is diagnostic if syphilis, caries, and necrosis of the nasal bones and foreign bodies are excluded. A sense of dryness is complained of. Occasional obstruction arises from accumulation of crusts, otherwise the passage is unduly open. There is constant hawking and spitting of brownish-green crusts, which are often blood-tinged. Frontal headache may occur in paroxysms. The patient is often depressed in spirit. The bridge of the nose may fall in slightly.

The Auxiliary Cavities of the Nose.—The Antrum.—The antrum is subject to abscess, cysts and polypi, tumors, and to the invasion of parasites.

Abscess.—An odor somewhat like that of ozena, a putrid taste, nausea, anorexia, pain in the cheek and at the root of the nose, often neuralgia in the frontal region, and malaise are present. A very characteristic symptom is the discharge of pus from one nostril on leaning the head forward. There is often a decayed tooth on the same side of the jaw.

Rhinoscopic examination shows a flow of pus into the middle meatus. Sometimes a probe can be passed into the antrum from the nose. When the foramen is obstructed, there is a dull aching pain in the upper jaw with deformity of the orbit, face, hard palate, and nostril. Fluctuation can usually be found at some point after a time. The x-ray shows a definite shadow in the antrum. Transillumination, *i. e.*, having the patient hold a small electric light bulb in the mouth while in a darkened room, will show light shining through the normal side only.

The Sinuses.—The frontal, ethmoidal, and sphenoidal sinuses are subject to inflammations, abscesses, traumatism, and the irritation of foreign bodies, usually parasites. The frontal sinuses are the only ones that exhibit external symptoms. When these cavities are inflamed the patient complains of pain and tenderness over the frontal protuberances; if the process goes to the formation of abscess, there may be redness and swelling and finally fluctuation. If the communication is not closed, a fetid discharge from the middle meatus takes place. Chronic frontal sinusitis is often the cause of chronic frontal headaches which are frequently misdiagnosed and called neuralgia.

When the sphenoidal and ethmoidal sinuses are affected, there are

DIAGNOSIS

The enlargement is so great as to affect the voice. Pain. Pus is seen coming into the mouth and backward into the pharynx. It may lead to abscess, necrosis, and necrosis. It is often the seat of inflammation by extension in the nose, and epiglottitis. On moving forward over the epiglottis, the meatus. On inspection, the ducts are found to be painful.

DISEASES OF THE LARYNX

about the cartilages, or perichondritis. During the course of an infectious disease. Diseases. The collateral edema is generally associated with cough, hoarseness, dysphagia. The larynx is extremely tender. The pain is increased by speaking or swallowing. Inflammation of the larynx characterized by hoarseness, with cough, hoarseness, and at times, the following are observed: (1) acute catarrhal laryngitis; (2) membrane laryngeal diphtheria; (3)

The causes are exposure to cold, the ingestion of hot liquids or corrosive substances, or from excessive use of the voice. It may be symptomatic of the eruptive diseases, such as erysipelas. The attack begins with fever of varying degree, but usually with a feeling of pressure and dryness in the throat, a tickling cough, dry, hacking, and hoarse. In severe forms the cough is almost continuous. When the patient speaks, or takes food, the cough is short time it becomes moist, and expectoration takes place, which may be yellowish, or may be lost entirely. A spasmodic symptoms occurs in acute laryn-

geal dyspnea occur in paroxysms, and are relieved by coughing up. The paroxysms take place at night, and may cause retching and vomiting.

(See page 504.)

Spasm False Croup or Spasmodic Croup). Spasmodic croup is seen associated with spasm of the larynx is seen

in children. The catarrhal symptoms are mild, so that the child seems to be well during the day. Fever is absent, and a slight cough or huskiness alone calls attention to the larynx. After the first three or four hours of quiet sleep the child suddenly awakes with a barking cough, sits up and struggles for breath. The dyspnea continues from a few minutes to an hour or so, gradually lessening, to disappear entirely as the child lapses into sleep. Throughout the next day the child seems well and the succeeding night is seized with another attack of "croup." This may occur once or twice during the night. It seems to be influenced by the weather. It recurs frequently during the same season.

EDEMA OF THE LARYNX.—This condition develops in the course of acute laryngitis, quite frequently in chronic diseases of the larynx, particularly if ulceration is present, in perichondritis and as a complication of erysipelas, typhoid fever, scarlatina, typhus fever, variola, and diphtheria. It may appear as a complication of acute phlegmonous inflammation of nearby structures or secondarily in the general edema of nephritis or cardiac disease, or from pressure of a thoracic aneurism. Secondarily, it occurs in angioneurotic edema and very frequently develops if foreign bodies become impacted in the larynx, or if irritating vapors are inhaled.

Symptoms of laryngeal stenosis may occur suddenly. The voice becomes husky and suppressed, the dyspnea is very extreme, so that in a few hours grave symptoms of obstruction arise. Death may occur unless the patient is relieved by tracheotomy. There is no cough. The patient complains of the sensation of a foreign body, and tries to grasp it. The swollen epiglottis can be seen or palpated with the finger.

Acute Submucous Laryngitis.—The inflammation extends to the submucous cellular tissue. It arises in the course of acute laryngitis, and is the form seen in traumatism, or from burns and scalds. The symptoms are those of intense laryngitis, with stridor. They increase in severity until stenosis arises. If the lower surface of the cords is affected death occurs from asphyxia. Sometimes the inflammation is circumscribed and is followed by the development of an abscess.

The chronic form of submucous inflammation of the larynx is usually seen in drunkards, and is recognized usually by the laryngoscopic examination. The symptoms are those of slight stenosis.

Chronic Laryngitis.—Chronic hoarseness may be due to chronic laryngitis. This affection either originates in an acute attack or comes on slowly. Prolonged use of the voice in a higher key than natural or in the open air, the abuse of alcohol, and constant exposure, are exciting causes. It may be symptomatic of syphilis or tuberculosis, and frequently results from inflammation of the upper air passages, and particularly from chronic pharyngitis. It occurs more frequently after middle life, usually in the male sex. Prolonged or excessive use of the voice gives rise to discomfort with dryness and tickling in the throat. At first the secretion of mucus is very slight, but after hawking and coughing it increases in amount. Hoarseness occurs, and if the patient

is careless or persists in the harmful occupation, complete aphonia may result. The voice is clearest in the morning, after expectoration of the nocturnal mucus, but becomes husky toward night. The aphonia may occur in paroxysms, and is relieved by coughing up a dry secretion. The cough is never severe. The sputum is small in amount, glairy, often in little balls or crusts.

Paralyses of the Laryngeal Muscles.—These are divided for convenience into groups. The chief symptom is alteration in the voice, which, with the laryngoscopic picture, leads to the recognition of the particular form of the paralysis.

1. **Paralysis of the Tensors of the Cord.**—The cricothyroid muscle is paralyzed; the superior laryngeal nerve which supplies the muscle is affected. The voice is deep and rough, and incapable of producing high tones. Usually the whole nerve is involved, and the result is anesthesia of the larynx and paralysis of the epiglottis.

Laryngeal Examination.—The epiglottis is fixed, and falls back against the tongue. The glottis forms a wavy line.

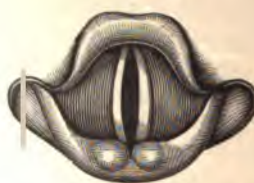
Causal Disease.—The condition occurs almost exclusively after diphtheria.

FIG. 181



Paralysis of the arytenoideus transversus in phonation. (Gottstein.)

FIG. 182



Paralysis of the thyro-arytenoideus internus in phonation. (Gottstein.)

2. **Paralysis of the Closers of the Glottis, or Adductors of the Cords.**—The muscles involved are the crico-arytenoideus lateralis, arytenoideus transversus, and the thyro-arytenoideus internus and externus. The nerve is the recurrent laryngeal.

The symptoms are complete aphonia, coming on suddenly, and often disappearing as suddenly.

Laryngeal Examination.—During phonation the cords remain in the inspiratory position. The paralysis may affect one or both sides. Sometimes the arytenoideus transversus alone is affected. Then there is hoarseness or aphonia. The anterior portions of the cords come together in phonation, but the posterior portions do not, leaving a triangular opening posteriorly (Fig. 181). In other cases the thyro-arytenoideus internus alone is affected. There is then dysphonia or aphonia, as before, but the cords come together at both extremities and remain apart in the middle, forming an oval opening (Fig. 182).

Causal Disease.—These paralyses occur in hysteria, catarrh, or severe overstrain of the voice.

3. Paralysis of the Openers of the Glottis, or Abductors of the Cords.—The muscle affected is the crico-arytenoideus posticus; the nerve is the recurrent laryngeal.

Symptoms.—When one side is affected, the respiration is free, but there is stridor, or forced inspiration. The voice is harsh.

Laryngeal Examination.—One cord remains in the median line. When both sides are affected, inspiratory dyspnea with stridor gradually develops. The voice is nearly normal. The glottis forms a narrow cleft which becomes still narrower on inspection.

Causal Disease.—(*Vide infra*—Paralysis of the Recurrent Laryngeal Nerve.)

4. Complete Paralysis of the Recurrent Laryngeal Nerve.—**UNILATERAL PARALYSIS.**—A weak, toneless voice which breaks into a falsetto when the patient endeavors to speak loud.

Laryngeal Examination.—The cord and arytenoid body are in the cadaveric position, viz., half-way between the phonating and the inspiratory positions. In phonation the other cord passes beyond the median line, and the glottis is slanting. The edge of the paralyzed cord is excavated.

BILATERAL PARALYSIS.—Aphonia and inability to cough. *Laryngeal Examination.*—Both cords are in the cadaveric position and their edges excavated. The adductors are usually paralyzed before the abductors, and one can see all the intermediate stages by close observation.

Causal Disease.—The conditions that give rise to the paralysis are numerous. It is due either to pressure on the vagus or recurrent laryngeal or some disease affecting those nerves or their roots.

The causes of pressure are: aneurism of the subclavian artery or aorta, mediastinal tumor, tuberculous bronchial glands, the apex of a tuberculous lung, cancer of the esophagus, goitre, and carcinoma of the pleura.

The diseases are: diphtheria, tumor, softening or hemorrhage into the brain, bulbar paralysis, neuritis, typhus, cholera, variola, articular rheumatism, sclerosis of the cord, progressive muscular atrophy, and general paresis.

Tumors of the Larynx.—Both benign and malignant growths are seen. At first dysphonia or aphonia takes place. Later dyspnea and dysphagia arise. These develop very gradually and in some few cases are attended by an irritative cough and the expectoration of fetid, frequently blood-stained material. The general symptoms, in the malignant forms, are pronounced, but cachexia develops later than in carcinoma elsewhere.

The *diagnosis* of malignant disease of the larynx is based upon the association of symptoms of laryngeal disease with pain, and with the characteristic appearances found on inspection, later associated with cachexia. Enlargement of the cervical glands points to cancer. Simple and syphilitic perichondritis must be excluded.

been shown to play a part in the causation of the condition. As with hay fever it is now supposed that essential asthma is in many cases a manifestation of an anaphylactic reaction.

Predisposing Causes.—The disease often manifests a distinct tendency to descend from one generation of a family to the next. Asthmatics are frequently neurotic and subject to other manifestations of their peculiar temperament. Changes in climate and season may cause the appearance of an attack. Males are more frequently affected than females (2 to 1). Diseases of the respiratory mucous membrane, particularly of the nose, are frequently liable to be followed by asthma.

Symptoms.—Premonitory symptoms, such as headache, neuralgia, irritability, vertigo, and drowsiness, occur in about one-half the cases. The attack usually begins during sleep and often at a regular time. The onset is manifested by tightness and feeling of constriction across the chest with some difficulty in breathing. The dyspnea increases rapidly and often reaches an extreme degree. The face is cyanotic and may be covered with a cold perspiration. The patient cannot get the air out of the lungs, and in his effort to do so calls into play all the accessory muscles of respiration. The breathing is extremely labored and difficult, yet the respiratory rate is much diminished sometimes to one-half the normal. The rhythm is also altered, inspiration being short and gasping, and followed without pause by a prolonged wheezing expiration. Subsidence of an attack is marked by expectoration, the sputum having special characteristics (see under Sputum). At first made up of mucous spirals, it later becomes mucopurulent. Curschmann's spirals and Charcot-Leyden crystals are nearly always found. Eosinophiles are found in the sputum, and they are always markedly increased in the differential blood count.

Physical Signs.—The chest is enlarged and rounded and the movements are lessened and strikingly out of proportion to the muscular exertion. On percussion hyperresonance is elicited; on auscultation expiration is faint and short, and inspiration is prolonged. Sibilant and sonorous rales are heard, more marked on expiration.

Sequela of Asthma.—The paroxysms of dyspnea may be present for years without any sign of changes in the lungs. In most cases, however, sooner or later there develops a *chronic bronchitis and emphysema*, which aggravate the original condition.

Diagnosis.—The sudden onset, the expiratory dyspnea, and the eosinophilia and other typical changes of the sputum, serve to differentiate the condition from acute or chronic bronchitis, tracheal or bronchial obstruction, and emphysema uncomplicated by asthma.

Bronchiectasis.—Dilatation of the bronchi occurs secondarily to affections which tend to weaken the walls of the tubes and to lessen their elasticity. Hence it is found in chronic bronchitis with emphysema, in chronic phthisis, in catarrhal pneumonia of children, in chronic obstruction from external pressure or foreign bodies. (See Obstruction.) It also occurs when the lung contracts in fibrous contraction.

pleural thickening. It occurs in two principal forms: the simple, in which the affected tubes are uniformly dilated; the saccular, in which larger or smaller pouches are formed. It is commoner in males than in females, and probably begins most frequently in adult or middle life. One lung only is affected in about one-half the cases, and when both lungs are affected (chronic bronchitis and emphysema) it is not often to the same degree.

Subjective Symptoms.—These consist of cough, expectoration, and a variable degree of dyspnea. Eventually there may be some loss of flesh. The cough is usually paroxysmal. It may occur only in the morning after the dilated tube fills and it may follow change in position. A paroxysm is followed by copious expectoration, sometimes amounting to a pint and a half in twenty-four hours. The sputum is grayish-brown and mucopurulent or purulent, faintly or extremely fetid, and contain pus and many microorganisms. In a conical glass the sputum separates into three layers: a frothy brown top, a thin mucoid layer in the middle, and a granular layer below. Hemorrhage may occur periodically even when tuberculosis is absent. Dyspnea is not usually severe except when the dilatation is complicated by disease of the heart or lungs, or by an acute attack of bronchitis.

Physical Signs.—The physical signs differ according to the extent and variety of the dilatation. In simple dilatation there may be nothing different from the signs found in chronic bronchitis, except a tendency to more bronchial respiration, with rales having a metallic quality. Percussion will vary according to the degree of alteration of the lung tissue surrounding the affected bronchi, and according to the extent of the dilatation and its proximity to the surface. In the simple forms the percussion note, if altered, is somewhat less resonant and higher in pitch; whereas in saccular dilatations favorably situated for percussion the note is tympanitic if the pouch is empty. On auscultation in simple dilatation the breathing approaches the bronchial and is accompanied by bronchial rales. In saccular dilatation the sounds are practically those of a cavity, respiration varying from bronchial to amphoric. Vocal resonance and tactile fremitus are usually both increased, but the latter may be diminished.

Diagnosis.—The diagnosis of simple dilatation from chronic bronchitis may be impossible, but copious and fetid expectoration indicates the former. The diagnosis of the saccular form from tuberculosis of the lung with cavity is difficult; but as cavities occur late in tuberculosis repeated search for the tubercle bacilli will soon clear up the diagnosis.

Bronchostenosis.—This may result from intrabronchial causes such as foreign bodies, fibrinous plugs, malignant tumors, syphilitic or tuberculous scars, etc., or from extrabronchial causes, such as pressure by enlarged mediastinal lymph nodes, mediastinal aneurism or tumor, or mediastinitis; tumors of the lung or esophagus; abscesses of vertbræ, sternum, or clavicles; effusions (pleural or pericardial), or by a greatly dilated left auricle.

Symptoms.—Sudden closure of a large bronchus causes marked dyspnea with circulatory disturbances and often rapid death. When closure is less complete, or more gradual, or the bronchus is smaller, there results atelectasis, compensatory emphysema, bronchiectasis, or bronchopneumonia. At times the obstruction acts like a ball-valve allowing the exit but not the entrance of air. A feeling of distinctly localized soreness is often present, but sharp pain is not characteristic.

Physical Signs.—These depend upon the size of the bronchus and the character of the obstruction. If the bronchus be small only the signs of the surrounding compensatory emphysema are detected with some increase in the respiration rate. If the bronchus be larger there is marked dyspnea and cyanosis, diminished expansion on the affected side, diminished or absent vocal fremitus, impaired resonance over the atelectatic portion, with a surrounding zone of hyperresonance, and diminished or absent breath-sounds over the atelectatic area. Over the site of the stenosis, sibilant and sonorous rales are audible, with at times a peculiar whirring sound that can even be felt as a thrill and which in some cases transmitted to the speech gives the voice a bleating character (Punch's voice).

DISEASES OF THE LUNGS

Acute Congestion (Hyperemia).—An increased amount of blood is forced into the lung, diminishing the air spaces, and causing small foci of hemorrhage. The inhalation of superheated air or irritating fumes, overaction of the heart and the initial stages of acute inflammatory disease of the lung may be the various factors causing the condition.

Symptoms.—*Sudden dyspnea, cyanosis, cough, and frothy, bloody expectoration* characterize the condition. Increased fremitus, impaired resonance, roughened breath-sounds, and subcrepitant or crepitant rales are found bilaterally at the bases of the lungs. If a primary condition, edema of the lungs may develop, otherwise the symptoms will disappear in a few days. If initial to a pneumonia of the lungs, the true condition soon becomes manifest with its appropriate symptoms and signs.

Passive Congestion.—*Mechanical congestion* occurs when the flow of blood to the heart is obstructed, as in organic valvular disease or relative insufficiency of the left heart. Rarely the pressure of tumors on the pulmonary veins acts in a similar manner.

Hypostatic congestion occurs in fevers, as protracted typhoid, and in prolonged general exhaustion or adynamia. Ascites or other affections below the diaphragm, which lessen the respiratory excursion, cause this form of congestion.

Symptoms.—*Dyspnea, cough, and expectoration of blood-stained sputum* are common. The sputum contains alveolar cells, often pigmented, "heart-failure cells."

Physical Signs.—Solidification is present, manifesting itself by slight dulness and feeble or bronchial breathing; the bronchial mucous membrane is also congested, giving rise to abundant large rales. The affection is bilateral and usually confined to the posterior portion of the bases.

Edema of the Lungs.—The transudation of serum into the air cells and alveolar wall occurs in all inflammatory and congestive processes of the lungs. It may be more or less general or in the immediate neighborhood of the disorder (inflammatory or collateral edema). *Acute edema* occurs: (1) in the course of diseases of the cardiovascular system, especially valvular disease, angina pectoris and arteriosclerosis; (2) in diseases of the kidney; (3) in certain infectious diseases; (4) in pregnancy; (5) in angioneurotic edema; (6) as an apparently primary condition, and (7) after paracentesis thoracis. The mechanism is probably best explained upon the theory that blood accumulates in the lung capillaries until transudation occurs, if there is disproportionate weakness of the left ventricle.

Symptoms.—The symptoms appear suddenly with substernal oppression, dyspnea rapidly going on to orthopnea, cyanosis and cough with copious expectoration of a thin white, often blood-tinged, frothy serous fluid. The patient's countenance is one of anxiety and the face is covered with sweat. The pulse is weak and rapid. Over the entire chest can be heard fine and coarse bubbling rales. The patient may die in a short time, "drowned in his own fluid," or the attack may pass away uneventfully in six to twenty-four hours.

Pulmonary Embolism, Thrombosis, and Infarction of the Lung.—The pulmonary artery or its branches may be plugged by an embolus, originating in the right heart the seat of an endocarditis or thrombosis, or in a vein, the seat of an already existing thrombus; the capillaries may be plugged by fat emboli.

Symptoms.—The occlusion of a large vessel by an aseptic embolus causes sudden intense dyspnea, cyanosis, and shock. The patient may die in a few moments or life may be prolonged for some hours. If a medium-sized vessel is obstructed, cough, hemoptysis, intense dyspnea, and symptoms of asphyxia rapidly develop. The heart action is irregular and weak. The patient passes into syncope and convulsions may precede death. Occlusion of a small vessel results in the formation of a hemorrhagic infarction. The symptoms are milder than in the first or second form and consist of dyspnea, palpitation, and hemoptysis. The quantity of blood expectorated varies from a large amount of bright red fluid to an occasional expectoration of rusty sputum. The spitting of blood may continue for several weeks, gradually abating as the infarction becomes organized and partially absorbed. Pneumonia and pleurisy frequently develop as complications. Rarely a *diffuse hemorrhagic infiltration of the lungs* develops in the course of hemorrhagic fevers, in pyemia and in certain acute cerebral diseases.

When the embolus is septic, the early symptoms are similar to the

more common with emphysema, but if the patient survives, pyrexia follows with a marked prostration of the lung disease.

Hemoptysis.—In the majority of cases after the onset of symptoms physical signs are absent. In the more prolonged cases, signs of pulmonary consolidation, often increased, tracheal, bronchovesicular or bronchial breathing, and crepitations and rhonchi are present usually in the lower lobes. A pleural friction rill may also develop and a vesicular blowing murmur may be heard over the heart and pulmonary artery.

Diagnosis.—The sudden onset of pyrexia and other pulmonary symptoms and the absence of a condition which would give rise to emboli, such as purpura, liver or vegetative heart disease, usually suffice to make a diagnosis.

Spontaneous Hemorrhage Hemoptysis.—Hemorrhage from the lung or bronchial tubes may occur in a number of conditions. The hemorrhage may be small in amount and may continue over a considerable period of time, or it may be sudden and profuse, at once terminating the life of the patient.

Hemoptysis may be due to (a) affections of the lungs, (b) affections of the lung.

Affections of the Lung.—1. *Congestion of the Lung.*—This may lead to hemorrhage. The amount of blood is small; it may be limited to discoloring of the expectoration, or it may be discharged in a few mouthfuls. This form of hemorrhage is seen in (a) organic heart disease; it is plus a characteristic feature of the first stage of (b) crupous pneumonia; in (c) hemorrhagic infarct hemorrhage occurs; in (d) phthisis it also occurs. (See notes.)

2. *Tuberculosis.*—In the very great majority of cases hemoptysis is caused by pulmonary tuberculosis. The hemorrhage may occur in tuberculosis either (a) as the first symptom of the disease, on account of colliquid congestion around infiltrated areas, or (b) later, on account of destruction through an artery after excavation of the lung has taken place. In the early stages the hemorrhage is usually profuse, but not fatal. It may recur repeatedly during a series of weeks. In the later stages the patient may have repeated hemorrhages, varying from a few ounces to half a pint or a pint. These may occur daily, or may be repeated at intervals of a week or more over a long period of time. The patient usually experiences much relief after the hemorrhages that occur at long intervals. Death rarely occurs after a large hemorrhage from a phthisical ulceration; yet it may possibly occur. Hemorrhage with the expectoration of calcareous masses may recur frequently (c) in patients with healed or quiescent tubercle.

3. *Cancer.*—In the absence of other causes, hemorrhage recurring frequently may be due to cancer of the lungs, especially if associated with currant-jelly sputum.

4. *Plastic Bronchitis.*—Hemorrhage is of common occurrence in plastic bronchitis when large bronchial casts are expelled.

5. *Gangrene*.—Pulmonary gangrene and in abscess of the lung hemorrhage occurs frequently, often causing death.

6. *Pulmonary Syphilis*.—Hemorrhage occurs relatively frequently in syphilis of the lung.

7. *Parasitic Affections*.—Actinomycosis, pulmonary distomiasis, etc., are extremely rare causes of pulmonary hemorrhage.

Affections Outside of the Respiratory Tract.—1. *Disease of the Blood-vessels*.—An aneurism situated close to the trachea and bronchi may rupture into these tubes, causing sudden profuse fatal hemorrhage. Sometimes for days the profuse hemorrhage is preceded by small hemorrhages. The physical signs of aneurism are sufficient to explain the cause. Endarteritis affecting the branches of the pulmonary artery is usually responsible for the hemorrhages that take place in the gouty aged of both sexes, independently of disease of the heart or of the parenchyma of the lungs.

2. *Cardiac Disease*.—In diseases of the heart, hemorrhages do not usually take place until secondary congestion of the lungs sets in. It may, however, be an early symptom in mitral stenosis. The hemorrhages may amount only to a staining of the sputum or to the expectoration several times during the day of an ounce or more of blood.

3. *Affections of the Blood*.—In these conditions hemoptysis is usually associated with hemorrhages in other portions of the body. Thus it may occur in hemophilia, in purpura, in scurvy, and in anemia. It also may occur in jaundice in connection with hemorrhages in other situations.

Hemorrhages from the lungs may be part of the blood dyscrasia of the severe infections, such as typhoid fever, hemorrhagic smallpox, typhus fever, and the like.

4. *Without Known Cause*.—*Disturbance of the Menstrual Function*.—Pulmonary hemorrhages occur in which it is difficult to find any cause. They are occasionally seen in females, sometimes at the menopause, in other cases during menstruation, in others during an interrupted menstrual period as vicarious menstruation.

Symptoms.—The only symptom of a small or moderate hemorrhage may be the presence of blood in the expectoration, or the spitting-up of a small amount of blood accompanied by a slight cough.

The symptoms of a large hemorrhage depend upon the amount of blood that is lost. Faintness and giddiness only may be present. When the symptoms are more pronounced, extreme pallor develops; the pulse becomes rapid, small, and feeble, the blood pressure falls; the extremities become cold, the face becomes bathed in perspiration; syncope may occur. If the patient recovers from the syncope, he is extremely restless, breathing hurriedly and sighing. There may be some nausea. Moderate delirium and mild febrile symptoms often follow. In the rupture of a large aneurism the blood rapidly wells up into the throat and pours out through the nostrils and mouth. With such hemorrhage the end may come in a few minutes. The blood from

SECRET

TO: DIRECTOR, FBI
FROM: SAC, NEW YORK
SUBJECT: [Illegible]
[Illegible text block containing several lines of typed communication]

RE: [Illegible]
[Illegible text block containing several lines of typed communication, including a reference to a letter from the New York office dated 1/15/54]

PLATE XI

FIG. 1

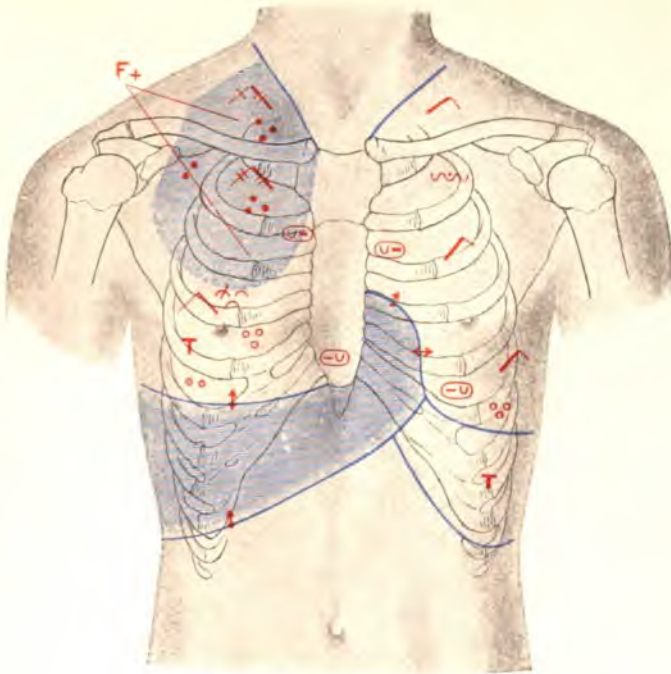
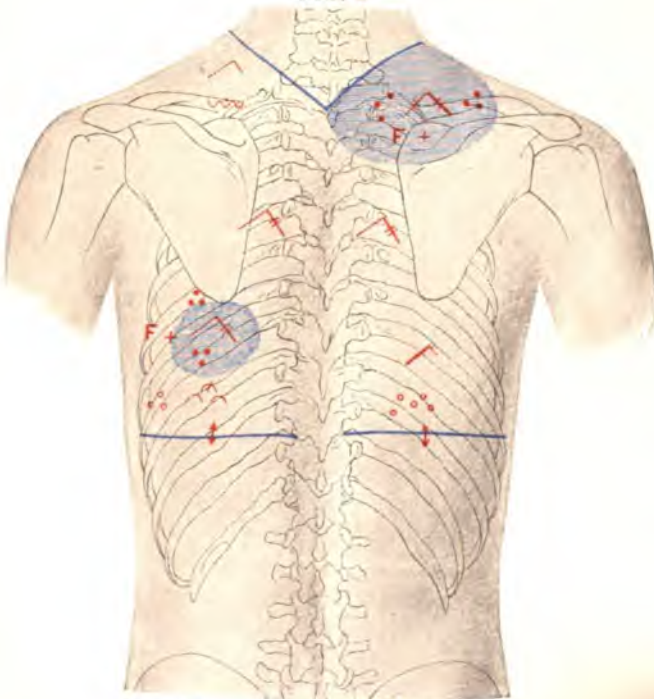


FIG. 2



Bronchopneumonia.

Consolidation in the right upper and the left lower lobes.



Fever develops, often beginning with a slight chill or a feeling of chilliness. The fever curve is irregular and the temperature persists usually two or more weeks, finally falling by lysis. The *pulse* is rapid and feeble. *Cough* and *expectoration* are marked. The cough is frequent, severe, and painful. The sputum is thick, glairy, and tenacious but not rusty. *Dyspnea* is pronounced and extreme. *Cyanosis* rapidly ensues, and is of a high grade in many cases, particularly in children who present the symptoms of asphyxia rather than those of the pulmonary involvement. In the aged the onset may be so insidious and the symptoms of pulmonary disease so mild that the actual lesions are only discovered by physical examination. Again the symptoms and findings may be obscured by a concomitant hypostatic congestion. In rare cases the *acute suffocative catarrh* of earlier writers develops so rapidly that in two or three days the patient may succumb to the overwhelming infection. The leukocytes are moderately increased unless there is severe and grave infection.

Physical Signs.—Early they are those of a bronchitis; later the signs of larger or smaller areas of solidification appear. Medium-sized coarse or fine mucus rales and slightly roughened breath-sounds are heard over the entire lung. The areas of solidification vary in size and location, an entire lobe may be more or less consolidated, or there may be only a few small isolated areas here and there. Over these, bronchial breathing, crepitant rales, increased fremitus and dullness on percussion are found. (See Plate XI.)

Diagnosis.—The affection is distinguished by remittent fever, extreme dyspnea and cyanosis, preponderance of physical signs of bronchitis over those of solidification, and long duration and gradual disappearance of symptoms. The disease must be differentiated from the *pulmonic form of acute miliary tuberculosis* by the absence of rapid emaciation, by the shorter course, by the more pronounced asphyxia, by the absence of tubercle bacilli in the sputum, and by the absence of signs of cavity formation.

Lobar pneumonia may be simulated by an acute primary bronchopneumonia, but in the former condition there is a continuous fever, less cyanosis, more pronounced leukocytosis, and the physical signs of solidification of one or more lobes without the diffuse bilateral involvement found in bronchopneumonia.

Atelectasis is differentiated by the absence of fever and by the fact that in bronchopneumonia the areas of dullness are subject to rapid changes in location.

Pulmonary Atelectasis.—Collapse of the lung may affect alveoli here and there or involve whole sections of the lung. Two forms are recognized, the *acquired* and the *congenital*. The acquired form is most often the result of occlusion of small bronchi and bronchioles by excessive mucus accumulating in the course of a bronchitis. Air cannot get into the vesicles on account of the blocking of the bronchiole and the air already present becomes resorbed, so that the vesicle collapses.

on infectious diseases. Tuberculosis of the lung, pulmonary phthisis, and consumption are names applied to a specific inflammation of the lung caused by the *Bacillus tuberculosis*. Clinically pulmonary tuberculosis may manifest itself in the form of (1) acute tuberculous pneumonia or bronchopneumonia, (2) acute miliary tuberculosis, (3) chronic ulcerative phthisis and fibroid phthisis.

Acute Pulmonary Tuberculosis.—THE PNEUMONIC FORM.—The disease may be secondary to an already existing chronic infection or appear apparently as a primary infection of the lungs. A single lobe of the lungs is usually involved, most often of the upper lobes. Adults are more frequently affected than children.

Symptoms.—The onset is acute, with *chills and a high fever*, which is continuous. *Cough, dyspnea, and cyanosis* are marked. The *pulse* is weak and rapid. The physical signs of solidification are found and the patient appears to be suffering from a frank pneumonia. Acute tuberculosis is, as a rule, not suspected unless the patient is known to have an existing tuberculous infection or until the crisis fails to occur and the fever is found to persist and becomes hectic. Emaciation develops rapidly. The tenacious, rusty sputum takes on a greenish hue and may become decidedly purulent in character. Tubercle bacilli and elastic tissue are found with the former as early as the fourth day. Hemoptysis may appear from time to time.

The *physical signs* have now become those of softening and cavity formation, usually associated with those of a dry pleurisy. Prostration is pronounced and the patient dies of asthenia in from two to twelve weeks.

BRONCHOPNEUMONIC FORM.—This form, the galloping consumption or phthisis florida of some writers, occurs most frequently in children, and is the usual form of acute pulmonary tuberculosis.

Symptoms.—These may develop suddenly, usually the result of aspiration of the contents of a tuberculous cavity into the finer bronchioles during hemoptysis, or the onset, though rapid, may be more insidious. A *cough*, at first non-productive, develops rapidly, with profuse expectoration of mucopurulent and at times bloody *sputum* laden with tubercle bacilli and containing elastic tissue. *Cyanosis, dyspnea, rapid pulse, and prostration* rapidly appear. The *fever* is irregular and hectic. The patient emaciates rapidly, and may die from six weeks to three months after the onset of the disease. Rarely the acute symptoms may subside and the condition becomes chronic.

Physical Signs.—At first are those of an acute bronchitis, but soon patchy areas of dullness appear, usually at the apex, together with harsh bronchovesicular or bronchial breathing and numerous coarse mucus and subcrepitant rales. Subsequently the area of dullness shows the physical signs of softening cavity formation.

Differential Diagnosis.—Tuberculous and non-tuberculous bronchopneumonia are often impossible to differentiate until tubercle bacilli are found in the sputum.

PLATE XII
FIG. 1.—Anterior Aspect.

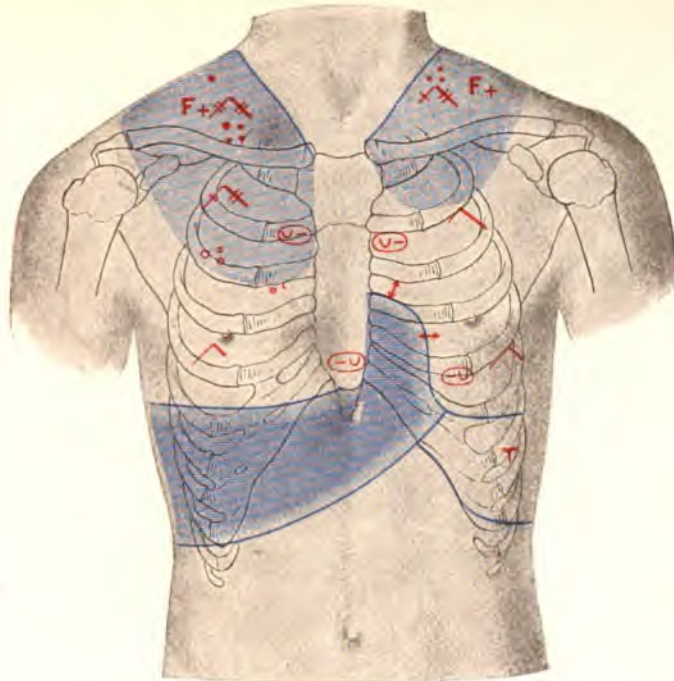
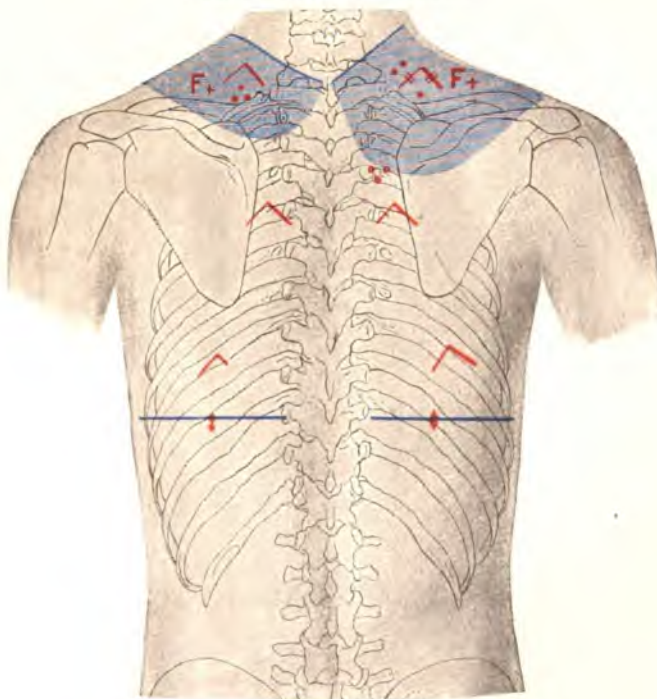


FIG. 2 —Posterior Aspect.



Acute Pulmonary Tuberculosis.

... of the left and



Acute Miliary Tuberculosis (Pulmonary Form).—The condition may develop in patients suffering from chronic tuberculosis of any organ, and frequently in children the extension of the disease is secondary to an attack of measles or pertussis.

Symptoms.—The onset is abrupt or may follow a period of malaise. *High fever, rapid emaciation, cough with mucopurulent, often bloody, sputum, hurried breathing, cyanosis, rapid pulse,* more or less stupor, delirium, prostration and the developments of the typhoid state are the characteristic symptoms. Gastro-intestinal or meningeal symptoms are frequent accompaniments of the more usual symptoms. Tubercles in the choroid can be at times demonstrated. Tubercle bacilli in the sputum are rarely found. A leukocytosis is usual. The duration of the disease is from two weeks to several months, always terminating in death.

Physical Signs.—These are not pronounced. Slight hyperresonance on percussion is usual. Diffuse crepitant or coarse mucus, sibilant, and sonorous rales are usually to be heard on auscultation.

Diagnosis.—The hurried breathing and cyanosis are the most distinctive local features of this disease.

Typhoid fever is differentiated by the presence of leukopenia, the Widal reaction, and the presence of typical stages in the course of the disease. *Septicemia* and *malignant endocarditis* are readily differentiated by the findings of the causative organism through the medium of a blood culture. The tubercle bacilli are found in the blood only rarely.

Chronic Tuberculosis.—Chronic Ulcerative Phthisis.—This is the usual form of pulmonary tuberculosis. The infection begins, as a rule, in the apices, at a point about 2 cm. below the extreme apex of the lung. From this primary focus it gradually spreads, by continuity to the adjacent structure of the original infected lobe, by inhalation, or by the lymph streams. The disease runs a more or less chronic course, often with periods of remission, during which the disease may be arrested temporarily or permanently.

Owing to the great importance of arriving at an early diagnosis the symptoms and signs of the *incipient stage* will first be described. Later the *moderately advanced* and the *far-advanced stages* will be discussed.

Symptoms.—The incipient stage is arbitrarily considered as that stage of the disease in which there is a slight infiltration of the apex or a small portion of one lobe without constitutional symptoms or tuberculous complications. The disease may begin in a variety of ways: the patient usually has been in poor health for some weeks, develops an ordinary bronchitis, with which pleurisy (chest pain) is occasionally associated (the commonest mode of origin). The cough proves obstinate, expectoration continues, and by and by the patient will be found to be losing strength and usually some weight. Examination will show now the physical signs of a slight apical involvement. In some cases fever, emaciation, and weakness or physical weakness alone

are evident for some time before the onset of pulmonary symptoms. Hemoptysis occasionally appears as the first symptoms or, more rarely, vague gastro-intestinal symptoms as epigastric distress, nausea and flatulence are the first manifestations of the disease. In the great majority of cases, however, *cough* is the initial symptom. As a rule only when the patient complains of a persistent cough is the disease in the incipient stage, and usually when other symptoms mark the apparent onset, the disease has progressed to a moderately advanced stage.

Physical Signs.—The early physical signs are not marked. On percussing over the apex there is found a small area of increased resistance with slight impairment of resonance as compared with the opposite side; the fremitus and vocal resonance are slightly increased or unaltered; the most characteristic findings are a slight increase in the transmission of the *whispered* voice-sounds and some prolongation of expiration, with a slight roughening of the expiratory sound. Such physical signs are met with most frequently immediately below the clavicle or in the suprascapular fossa. A few fine crackling rales may at times be heard at the end of inspiration, particularly if the patient is made to cough and then inspire deeply.

Diagnosis.—The diagnosis of the incipient stage of tuberculosis is most difficult. The history and clinical symptoms are of primary importance; more so than are the physical signs. The occurrence of cough and expectoration, progressive weakness or loss of weight, persisting for some weeks without apparent cause, in a person exposed to infection, even when the physical signs are but slight, is usually sufficient data upon which to base a diagnosis. As aids in the diagnosis we may have recourse to the various tuberculin reactions (see page 495) which often yield valuable information. The observation of slight variations in the patient's temperature may also be of service. The temperature should be taken every two or three hours for at least a week; a slight persistent rise in the temperature in the afternoon or evening or after exercise is usually indicative of a tuberculous lesion. The blood-count usually shows a slight secondary anemia and a leukopenia with an increase of the small mononuclear cells. Tubercle bacilli are very rarely found in the sputum in the incipient stages. The x-ray is a most worthy adjunct to the carefully studied clinical history and physical findings.

MODERATELY AND FAR-ADVANCED STAGES.—The cases that present more marked constitutional symptoms and signs than those above described, are arbitrarily considered in the moderately advanced stage of the disease. Those cases that present signs of intense solidification, softening with cavity formation, marked impairment of local and constitutional function and serious tuberculous complications, are considered in the far-advanced stage of the disease.

Symptoms.—The difficulties in diagnosis are now replaced by signs and symptoms that are characteristic and unmistakable. The pulmonary symptoms of the incipient stage persist, but with greater vigor and more marked reaction. The *cough* is frequent, often severe, and

PLATE XIII

FIG. 1.—Anterior Aspect.

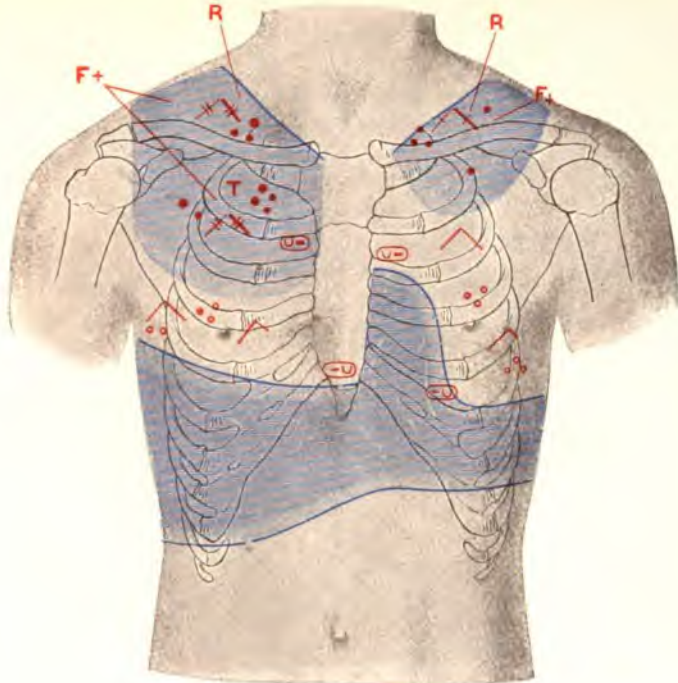
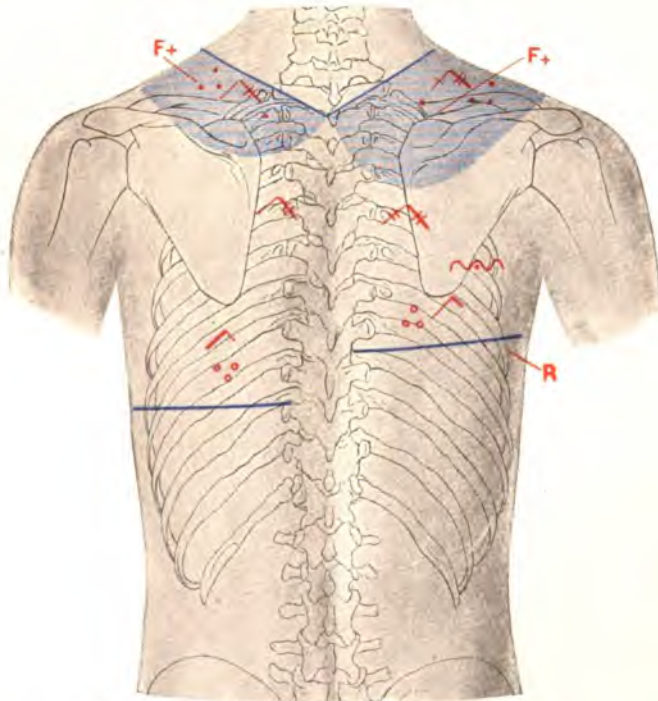


FIG. 2.—Posterior Aspect.



Chronic Pulmonary Tuberculosis.

dation with cavity formation. Chronic pleurisy with loss of resj



with more or less profuse *expectoration* of greenish-yellow sputum, which contains elastic tissue and tubercle bacilli. (See Sputum.) Nummular sputum is common in phthisical excavation, and this term describes round, tough masses of sputum which sink in water. *Dyspnea* may occur only upon exertion, but in the last stage is usually constant. *Cyanosis* gradually becomes marked, the fingers particularly are dusky and become clubbed. *Chest pain* is due to a localized pleurisy. It tends to recur, is unilateral, and often is not accompanied by the physical signs of an ordinary inflammatory pleurisy. *Hemoptysis* is usually due to the erosion of a vessel into a cavity. The amount may vary from a few cubic centimeters to a quantity sufficient to exsanguinate the patient.

In addition to the intensified pulmonary symptoms, the symptoms of the constitutional reaction now puts in an appearance; *emaciation* is a marked symptom, always found if the disease is becoming progressively worse. *Anemia* is pronounced and a leukocytosis is found if there is cavity formation. *Fever* is always present and may be intermittent or remittent, usually changing irregularly from one type to the other. The greatest variation in the range of the temperature is found in the far-advanced stage when there is a cavity with mixed infection. *Profuse sweats* may take place in the early stages, but are more usual in the far-advanced stages, occurring characteristically during sleep, the so-called "night sweats."

Gastro-intestinal symptoms of anorexia, vomiting, and diarrhea are usually the result of secondary infection of the gastro-intestinal tract in the advanced stages. *Vomiting* frequently follows a severe paroxysm of coughing. The *pulse* is one of fair volume and poor tension. The rate is increased, rising markedly with the fever, and is an important concomitant finding.

Physical Signs.—It is of extreme importance to always compare both sides in eliciting the physical signs of pulmonary disease, remembering the variations in the physical signs that normally occur in the lungs as a result of anatomical differences.

Inspection shows a long flat chest with depression of the supraclavicular and infraclavicular spaces and atrophy of the suprascapular muscles on the affected side. *Palpation* shows deficient expansion of the affected side with an increase of tactile fremitus over areas of solidification and cavities. Pleural thickenings or effusions frequently obscure the fremitus.

Percussion.—Over solidified areas the note is dull. The degree of dullness varies with the width and depth of the solidification; both light and dull percussion with deep and shallow breathing should be employed in questionable cases. Over small areas of infiltration the surrounding emphysema will often cause only a hyperresonant note to be elicited. Over cavities a hyperresonant note is the rule, though distinct tympanitic or amphoric resonance may be obtained over a large superficial cavity. Other phenomena, such as Wintrich's or Gerhardt's change of sounds and the "cracked-pot sound," may also be

elicited. Determination of the size of the lung and of the extent of the expansion is important. Percussion over the apices will demonstrate retraction or change in size at that point by a diminution in the areas of resonance or modified dulness. Percussion at the bases will mark the lowest boundary of the lungs, and at the same time by having the patient breathe deeply the extent of respiratory excursion can be determined, as in tuberculosis it is decreased or entirely absent on the affected side.

Auscultation.—The character of the respiratory murmur ranges for a simple prolongation of expiration, through bronchovesicular up to bronchial breathing, according to the extent of the solidification. Over cavities cavernous and amphoric breathing is heard. Crepitant and subcrepitant rales are heard over areas of solidification. They are more marked at the end of inspiration, and at times can only be elicited after the patient coughs and then inspires deeply. Rales of all varieties may be heard diffusely over the chest, the result of an accompanying bronchitis. Sibilant, sonorous, and coarse mucus rales are heard over cavities. The vocal fremitus is increased and whispered and spoken pectoriloquy and bronchophony may be elicited over areas of well-marked solidification as well as over cavities.

Other phenomena found at times are pleural friction rubs, cardio-respiratory murmurs, and systolic murmurs in one or both subclavian arteries.

Diagnosis.—The diagnosis of advanced tuberculosis is usually readily made. A positive diagnosis is obtained by finding tubercle bacilli in the sputum and in suspected cases repeated examination should be made. Other aids are discussed under diagnosis of incipient tuberculosis. The mistakes in diagnosis that do occur are the result of insufficient physical and other examinations. Advanced tuberculosis would not so frequently be called malaria, anemia, chronic bronchitis or cancer if a careful study of suspected cases were made.

FIBROID PHTHISIS is a form of chronic tuberculosis characterized by the formation of extensive fibrous tissue in the affected portion of the lung. It runs an extremely chronic course and is only differentiated from chronic interstitial pneumonia by the finding of tubercle bacilli in the sputum.

Gangrene of the Lung.—Gangrene is a rare disease of the lung and always secondary to other diseases. It may be circumscribed or, more rarely, diffuse. It results most frequently from pneumonia but may be due to injury, embolism, bronchopneumonia from aspiration of partic of food (in the insane), bronchiectatic and tuberculous cavities, or may arise in the course of diabetes, or follow or complicate general septic conditions.

Symptoms.—There is moderate fever, prostration, hemoptysis, a with the expectoration of profuse brownish purulent sputum which has a fetid, penetrating, persistent, and often sweetish odor. It contains

fragments of lung tissue, altered blood, and putrid debris, and separates into three characteristic layers when placed in a conical glass. The fetor of the breath and of the characteristic sputum are diagnostic.

Abscess of the Lung.—The symptoms are those of a cavity associated with a diffuse bronchitis. A pulmonary abscess is always secondary either to intra- or extrapulmonary disease. If the former it may follow an acute inflammatory process of the lung (lobar, lobular, and especially aspiration pneumonia) or result from the action of the tubercle bacilli. Extrapulmonary causes are traumatism, infective emboli, usually pyemic, and rupture of an abscess outside the lungs into the pulmonary tissue.

Symptoms.—If the abscess is of sufficient size there will be found irregular *chills and fever*, *cough* associated with *purulent sputum*, which may be profuse and continuous or profuse only at irregular intervals, *i. e.*, when the abscess cavity is emptied by paroxysms of coughing or by change of position. The sputum is often inoffensive and contains particles of lung tissue and elastic fibers. If there are many small pyemic abscesses the symptoms are those of a pyemia, and the local signs are frequently masked or overlooked.

The *physical signs* of a large abscess are those of a cavity which is intermittently filling and emptying.

Differential Diagnosis.—*Pulmonary gangrene* is more acute in onset, runs a more rapid course, and has a characteristic offensive putrid expectoration.

A *tuberculous* cavity may present similar signs and symptoms, but as it is a late manifestation of tuberculosis the tubercle bacilli can be found by sputum examination.

Bronchiectasis runs a chronic, pulmonary abscess an acute course.

Pulmonary Emphysema.—Emphysema is due to atrophy of the walls with permanent distention of the air vesicles. An increase of intra-alveolar air pressure with possibly a congenital defective development of the pulmonary elastic tissue is necessary for the development of the pathological changes. It occurs most frequently in children, the result of nasal and nasopharyngeal obstruction or from hereditary predisposition, and in the aged, the result of chronic bronchitis, asthma or other chronic diseases of the respiratory tract and, to a lesser extent, chronic cardiac disease, pleurisy with adhesions of the two layers of the pleura, or it may be due to the rigidity of the costal cartilages with consequent loss of the elasticity of the thorax. The influence of occupation (glass-blowing, playing on wind instruments, etc.) seems to be negligible.

Symptoms.—*Dyspnea* and *cyanosis* are the prominent symptoms. The *dyspnea*, expiratory in character, is in proportion to the degree of emphysema, and is aggravated by the coexistence of bronchitis, asthma, and eccentric hypertrophy of the right ventricle—frequent complications in cases of long standing. In the later stages of the disease the *dyspnea* is constant and extreme. *Cyanosis* is marked; the

lips and finger tips are bluish and the face a dingy pale color. *Cough* varies in frequency, but sooner or later becomes a constant concomitant of the condition and is the natural sequel of frequent attacks of bronchitis which eventually terminate in a persistent bronchitis. The cough, before it becomes chronic, is worse in winter and often disappears entirely in summer.

The expectoration is that of chronic bronchitis. The general health suffers by loss of strength and capacity for physical and mental work, rather than by loss of flesh.

Physical Signs.—(See Plate XIV.) The patients are large-chested and stoop-shouldered, with a continual aspect of anxiety. In well-marked cases the chest is *barrel-shaped* (see Inspection). There is little movement of the chest during respiration and the expiratory effort is prolonged. Both tactile and vocal fremitus are decreased. The percussion note is hyperresonant, often slightly tympanitic. The normal limits of pulmonary resonance are uniformly increased. Auscultation will characteristically show a weakness of the vesicular murmur, which is low-pitched both in inspiration and expiration. The relative length of inspiration and expiration is usually about equal, although expiration may be even longer than inspiration. Sibilant and wheezy rales, heard diffusely over the chest, as well as coarse and fine crackling and bubbling rales, are due to the accompanying bronchitis.

Cardiovascular Signs.—The pulse is weak but slow. The veins of the neck are prominent, perhaps pulsating; the apex-beat cannot be seen nor felt. The area of heart dulness is diminished or even absent, and the heart is pushed over to the right. The *heart-sounds* appear feeble and distant. The right ventricle becomes hypertrophied and ultimately dilated. The *pulmonary second sound* is accentuated. A tricuspid regurgitant murmur may be heard. Venous congestion, albuminuria, and edema of the feet and legs may occur in the later stages.

Diagnosis.—This is based upon the chronicity of the condition, the occurrence of dyspnea and cyanosis, and the physical findings. Emphysema may be differentiated from *chronic bronchitis* by the hyperresonant note on percussion and the prolongation of expiration on auscultation.

Pneumothorax develops suddenly, affects one side, and gives a distinctly tympanitic percussion note and an absence of true vesicular breathing.

Pleural effusion is unilateral and the percussion note is flat.

Varieties of Emphysema.—The above form, substantive or hypertrophic emphysema, is the one which is clinically recognized as emphysema when that term is used. Other forms of emphysema are recognized but differ pathologically and clinically from true emphysema.

Senile or atrophic emphysema is a true atrophy of the lungs. The condition is found in the aged. The thorax is small, narrowed, and with lessened respiratory excursion.

PLATE XIV

FIG. 1.—Anterior Aspect

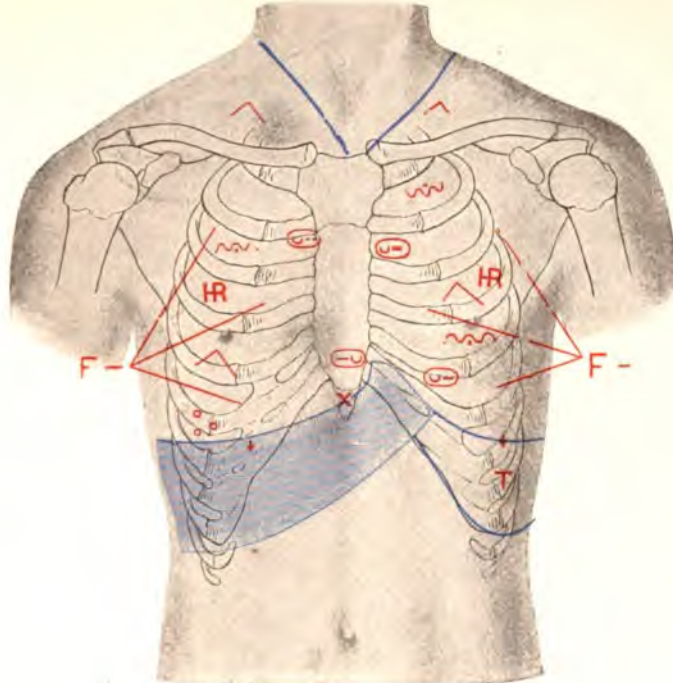
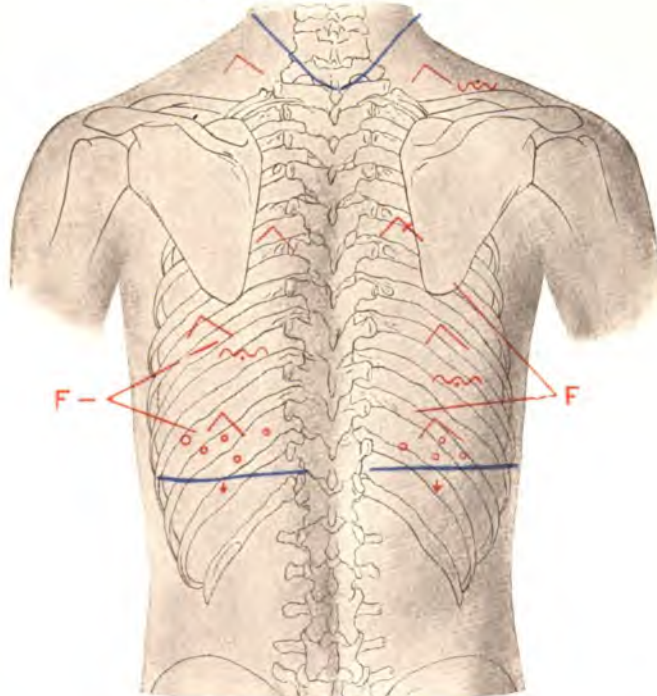


FIG. 2.—Posterior Aspect.



Emphysema.

Hyperresonance. Enlargement of lungs and diminished respiratory movement.



Compensatory emphysema is the result of an unaffected portion of the lungs vicariously taking on the function of another portion of the lungs which is not functioning. The condition may occur locally, as around small areas of bronchopneumonia or tuberculous infiltration and cavity, or it may be general when one lobe is crippled by a pneumonia, a pneumothorax, an atelectasis, or a large pleural effusion. The physical signs when demonstrable are those of substantive emphysema.

Acute vesicular emphysema is a rapidly developing overdistention of the air vesicles occurring in asphyxia, asthma, angina pectoris, or pertussis. It is not a true emphysema, as recovery or death ensues before atrophy of the elastic tissue takes place.

Interstitial emphysema is characterized by the presence of air in the interlobular and subpleural tissues. It is due to wounds of the lungs or rupture of the air vesicles during violent coughing. The condition usually is not recognized unless a subcutaneous emphysema in the neck or a pneumothorax develops.

New Growths in the Lungs.—Cancer of the Lungs.—The new growth may be primary or secondary. The latter is most common. Secondary new growths succeed disease in the abdominal organs, the genito-urinary tract, the bones, the breast, and the eye.

Symptoms.—The general symptoms of carcinoma accompany the thoracic symptoms. *Chest pain, dyspnea, cough,* and a peculiar expectoration belong to the latter. The pain is due to associated pleurisy; dyspnea is paroxysmal. (See Dyspnea from Pressure on Bronchi.) The *expectoration* is dark like prune juice. Signs of intrathoracic pressure are seen. The external thoracic veins are enlarged. The face and arms may be cyanosed, or one arm only may be affected. The heart may be dislocated; compression often by mediastinal glands, of trachea and bronchus causes dyspnea, of the esophagus, dysphagia.

Physical Signs.—In primary cancer the affection is unilateral; in secondary forms, bilateral. The *physical signs* are those of *pleural effusion* or of local *solidification*. The solidification may be massive and not partake of the shape of a lobe. Often signs of effusion and solidification are combined (enlargement, immobility, absent fremitus, but bronchial breathing). In the secondary forms the disease is bilateral. The signs are mixed. They indicate diminished air in the lung structure. Care must be taken not to overlook the pleural effusion which accompanies the process, the removal of which gives temporary relief. In both forms external lymphatic glands, particularly the cervical, may be enlarged.

Diagnosis.—The diagnosis is based upon (1) the age (after forty); (2) the occurrence of emaciation; (3) the duration of the disease, often rapid, rarely beyond eight months; (4) the presence of primary disease elsewhere; (5) the presence of moderate fever; (6) the signs of intrathoracic pressure; (7) the involvement of lymphatic glands; (8) the occurrence of irregular areas of solidification and of pleural

effusion, alone or combined; (9) the characteristic expectoration; (10) dyspnea due to pressure on the bronchus or trachea; (11) the absence of bacilli from the sputum.

An effusion can often be recognized only after puncture. Hemothorax is often but not necessarily present.

Sarcoma.—This is sometimes primary, but more often secondary. The *symptoms* and the *physical signs* are much the same as in carcinoma. The age (usually under forty) may be an aid in *diagnosis*.

Gross Parasites of the Lungs.—**Hydatid Disease of the Lungs.**—The lungs are affected in about 11 per cent. of the cases of hydatid disease.

Symptoms.—According to Wilson Fox, these consist of *dyspnea*, *pain in the chest*, *cough*, occasional *hemoptysis*, and sometimes the *expectoration* of hydatids, the *sputum* being otherwise bronchitic, or presenting the characteristics of pneumonia or gangrene when these complications are present. Gradually *weakness* increases, sometimes with *pyrexia*, which, when combined with *emaciation*, may impart to the case a considerable resemblance to phthisis. *Pressure symptoms* occasionally occur.

Physical Signs.—These are either those of *solidification* of the lung or of *pleural effusion*, together with certain peculiarities depending on the size and site of the tumor.

Diagnosis.—The symptoms present—cough, dyspnea, anemia, emaciation, and clubbing of fingers—too often lead to diagnosis of *phthisis*. Hemoptysis occurs in many cases. The temperature is normal, an important point in diagnosis. If the cyst ruptures, the sputum is diagnostic. Complications often mask the diagnosis. It must be distinguished from *pleurisy*, localized *empyema*, *pulmonary abscess*, *phthisis*, *actinomycosis*, and *mediastinal tumors*.

DISEASES OF THE PLEURA

Pleurisy or Pleuritis.—This is an inflammation of the pleura. The exciting cause is always some microorganism, although what variety it is cannot always be determined. Predisposing causes are exposure to cold or trauma; more frequently it occurs secondarily to (1) diseases of the lung, as pneumonia, tuberculosis, gangrene, or abscess; (2) extension of inflammatory disease of the ribs, vertebræ, pericardium, mediastinum, aorta, and other neighboring structures; (3) diseases below the diaphragm, as acute hepatitis, splenic or pancreatic abscess; (4) general disease, as septicemia, acute rheumatism, and scarlatina.

Acute Fibrinous Pleurisy.—Dry or plastic pleurisy may result from any of the above causes. It almost constantly accompanies lobar pneumonia and gangrene or abscess of the lung if the inflammatory processes reach the periphery of the lungs.

Symptoms.—The characteristic subjective symptom is *pain in the side*, usually on a level with the nipple. It is sharp, cutting, or tearing

PLATE XV

FIG. 1.—Anterior Aspect.

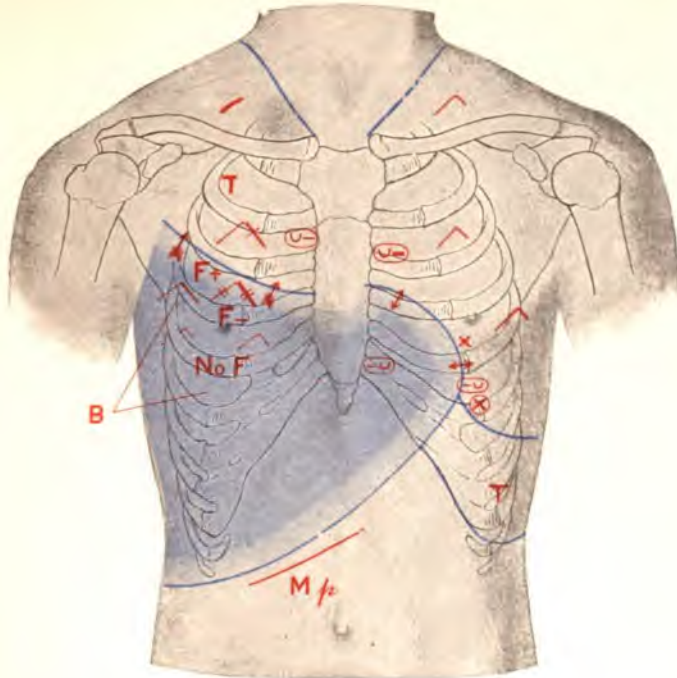
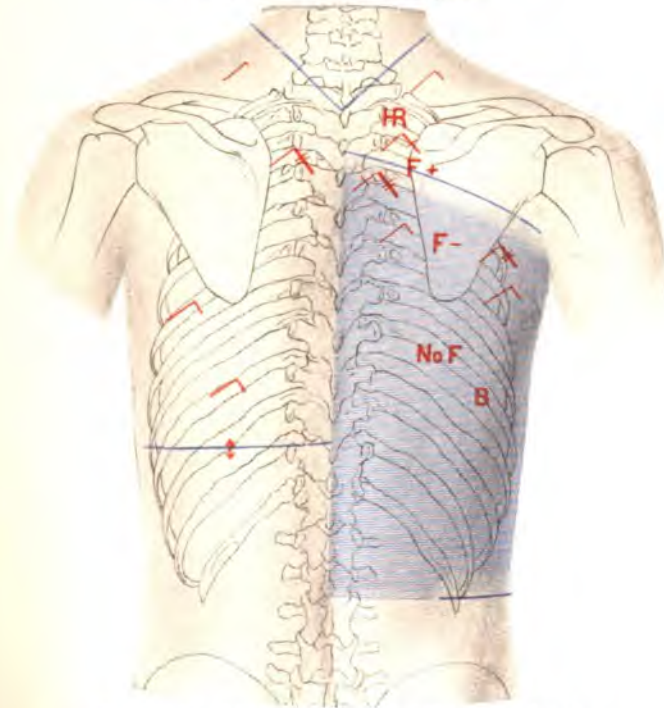
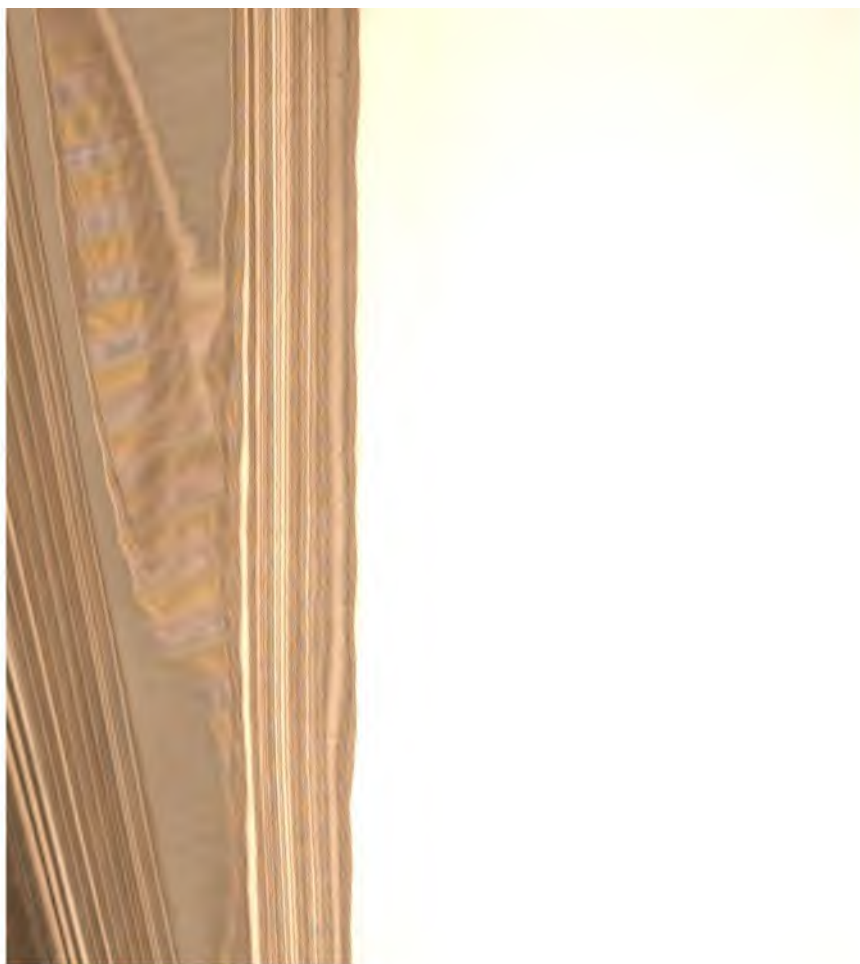


FIG. 2.—Posterior Aspect.



Pleurisy with Effusion (right-sided).



in character, exaggerated by breathing or coughing and frequently referred to the seat of the pleurisy. The patient restricts respiratory excursion by leaning toward the affected side, and thus lessens the pain. *Cough*, dry and painful, is present. The *breathing* is hurried, shallow, irregular, and chiefly abdominal in type. Fever of a mild degree is usually present.

Physical Signs.—Distinct *limitation of movement* of the affected side is seen. A *friction rub* (see page 319) is heard usually over the seat of pain.

Differential Diagnosis.—*Intercostal neuralgia* and *pleurodynia* are usually readily differentiated from pleurisy by the absence of fever and a friction rub. Moreover in the former condition there is tenderness over the exit of the intercostal nerves and in the latter, pleurodynia, the pain is exaggerated by twisting or turning the side as well as by deep breathing.

Serofibrinous Pleurisy.—Pleurisy with effusion is usually preceded by the symptoms of a dry pleurisy, but may develop apparently without the intervention of those symptoms.

In a great majority of cases, it is due to a tuberculous infection. Other causes are those that have already been enumerated.

Symptoms.—There occurs slight or moderate *fever* sometimes intermittent in character, with recurrent chills, and terminating by lysis in one to three weeks; considerable *dyspnea* occasionally amounting to *orthopnea* when the effusion is very extensive; and a dry *cough*. There is frequently some evidence of slight *cyanosis*. *Pain*, similar to that of a fibrinous pleurisy, is often seen when the effusion is small, but when it has become of any size it is characteristically absent, as the inflamed layers of pleura are prevented from rubbing against each other by the excessive fluid. The urine is decreased in advancing effusions but increases in amount with decline of the fluid. The leukocytes are unchanged in the majority of cases.

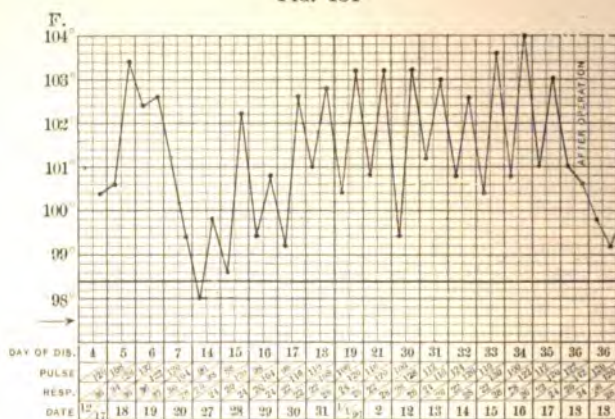
Physical Signs.—(See Plate XV.) There is seen *enlargement of the affected side*, increase in semicircumference with fulness of the interspaces, and diminution of movement. Vocal and tactile fremitus are diminished or absent, although the whispered voice is heard better through a serous than a purulent effusion (Bacelli's sign).

There is dulness or flatness on percussion with great increase to resistance of the pleximeter finger. While above the effusion the percussion note is hyperresonant or tympanitic (Skoda's resonance). In medium-sized effusion an S-shaped line of dulness can be demonstrated as the upper limit of the effusion. The line begins rather low down at the spine, curves upward and obliquely across the chest into the axilla, whence it descends to the sternum. Movable dulness may be demonstrated with changes in the position of the patient. Triangular dulness may be found on the opposite side of the effusion. The base is at the level of the lower border of the lung and the apex about the height of the upper level of dulness (Grocco's sign). The presence of a normal

triangular area of paravertebral dulness diminishes the diagnostic value of this sign. The lower border of flatness is below the normal lung limits. If there is marked compression of the lung bronchial breathing may be heard. Egophony, at the upper border of the effusion, and bronchophony, over a large effusion, can at times be made out. Above the effusion the breath-sounds are bronchovesicular or bronchial, and a friction rub or fine rales may be heard.

Displacement of Organs.—If the effusion is on the left side the heart is displaced toward the right and Traube's semilunar space is often entirely effaced. A right-sided effusion pushes the diaphragm and liver downward and displaces occasionally the heart more to the left than normal.

FIG. 184



Empyema following pneumonia. (Fever absent from seventh to fourteenth day.) (Original.)

Purulent Pleurisy.—Empyema.—This term is applied to pleural inflammations characterized by pus formation.

It results most frequently as a sequel of pneumonia, bronchopneumonia, or a serofibrinous pleurisy; more rarely it occurs in the course of pulmonary tuberculosis; as a result of a penetrating wound of the pleura; during an attack of scarlatina; or it may result from a rupture of a subphrenic abscess into the pleural cavity.

Symptoms.—The condition is commonly insidious in onset with the general symptoms of sepsis overshadowing the local symptoms of slight cough, with little or no expectoration, mild dyspnea and moderate pain. The fever is high, usually extremely irregular, and attended by chills at the beginning and sweats at the end of the febrile paroxysm. Pallor, weakness, and prostration develop rapidly; a marked leukocytosis is present. Indicanuria and albuminuria will be found. Erosion of the chest wall and escape of pus are indicated by the presence of a subcutaneous fluctuating inflammatory tumor—*empyema necessitatis*. It

usually found in the fifth space, anteriorly, or below the angle of the scapula, posteriorly.

Physical Signs.—The physical signs of empyema are those of other effusions within the pleura. In addition there may be present more marked *bulging of the affected side*, *local edema* of the chest wall and distinct *bronchial breathing* transmitted along the chest wall. *Bacelli's sign* (*v. s.*) is a valuable finding.

Hemorrhagic Pleurisy.—In this rare condition the bloody exudate may be due to malignant disease or tuberculosis involving the pleura; to malignant and hemorrhagic forms of acute infectious disease; and to disease of other organs, as liver and kidney, associated with pronounced vascular changes. The condition must be differentiated from hemothorax due to rupture of a vessel or an aneurism within the pleural sacs.

Other Varieties of Pleurisy.—**Diaphragmatic Pleurisy.**—This may be either dry or with effusion, may affect only the diaphragmatic pleura, or may be a general pleurisy. There is intense *pain in the epigastrium* and often extending from the anterior end of the tenth rib to the ensiform cartilage. The pain is aggravated by breathing, which is thoracic, rapid, and feeble in type. *Tenderness* may be elicited over the phrenic nerve in the neck. Other occasional symptoms are *nausea*, *vomiting*, and *obstinate hiccough*. The *fever* is usually higher than in ordinary pleuritis. Effusion may lessen the pain. Peritonitis may occur at the same time or be secondary to the pleurisy. Litten's phenomenon is absent (see page 301).

Interlobar Pleurisy.—In the interlobar form, *interlobar pleurisy* as a result of a general pleuritis, the two layers of pleura between lobes at times become partially adherent, enclosing collections of fluid either serous or purulent. If the latter and more frequent condition, the diagnosis is usually impossible unless the pus ruptures into a bronchus or unless the aspirating needle can disclose the presence of the pus.

Pulsating Empyema.—A localized pulsating collection of pus moving synchronously with ventricular systole of the heart may be mistaken for an aneurism. The pulsation is detected by inspection and palpation. It may be confined to two or three interspaces or occupy the anterior aspect of the thorax and axilla of the left side. Rarely the pulsation is posteriorly, and it is practically always on the left side. The presence of fever and a leukocytosis, the absence of murmurs and expansile pulsation, and the fact that the tumor decreases in size and tension on deep inspiration, are findings sufficient to differentiate a pulsating empyema from an aneurism.

Tuberculous Pleurisy.—This may give rise to either a fibrinous or serofibrinous pleurisy. Frequently it results in marked thickening of the pleura.

Diagnosis of Pleurisy with Effusion.—The diagnosis is based upon the physical signs. Atypical cases sometimes simulate *croupous pneumonia*. In doubtful cases resource may be had to the exploratory

needle (see page 440). If the variations in the physical signs are not sufficient to establish a diagnosis, aspiration with a large sized hypodermic needle will yield serum if there is a pleural effusion, a few drops of dark blood if pneumonia.

Hydrothorax is differentiated by the absence of fever, pain, and friction rub, by the character of the aspirated fluid and by the concomitant presence of heart or kidney diseases.

Pericardial effusions, if large, are differentiated by the absence of dullness at the base of the lungs, by the convex area of dullness to the right of the sternum, by the absence of cardiac impulse, and by the pronounced dyspnea and cardiac weakness, not observed in pleural effusions of a size sufficient to simulate pericardial effusion.

Intrathoracic Tumors.—There are present symptoms of pressure on contiguous structures, irregular outlines of dullness and enlargement of lymph glands with signs of malignancy in other parts of the body since pulmonary new growths are most frequently secondary to new growths elsewhere in the economy. In many cases, however, pleural effusions occur as a result of new growths in the thorax, so that an absolute differential diagnosis of the condition is often impossible until pressure symptoms and cachexia are marked.

Subphrenic abscess is differentiated from an empyema by the presence of respiratory movements of the lower border of the lung. In a similar manner, *abscess, hydatid cysts, and cancer of the liver* are differentiated from pleural effusions. In addition these conditions show a convex line of dullness at the right base of the lungs over which a friction rub can often be heard.

Purulent effusions can be diagnosticated and usually definitely localized by means of the x-ray.

Data Obtained by Paracentesis Thoracis.—Aspiration of the pleural cavity yields information which renders valuable aid in the differentiation of the conditions causing pleural effusions and positive information of the character of the effusion. Pus needs no description. Hemorrhagic and chyliform fluids are recognized as soon as withdrawn. Exudates, the result of inflammatory processes, and transudates are usually readily differentiated by appropriate laboratory procedures (see page 447). If serous fluid is withdrawn and is shown to be inflammatory in character the injection of this fluid in large amount into animals will usually result in an accurate determination of the causative organism. Cultural methods of isolating the organisms are not satisfactory in most cases, owing to the small number of organisms in the fluid. Fragments of cancerous tissue may occasionally be found in malignant disease of the pleura.

Chronic Pleurisy.—This may be manifested by an adhesive form or associated with effusion. *The dry or plastic form is the result of an acute attack of serofibrinous pleurisy in which the fluid has been resorbed or aspirated; it may follow a chronic pleurisy with effusion; at times it occurs as a primitive condition, which is followed by pulmonary*

PLATE XVI

FIG. 1.—Anterior Aspect

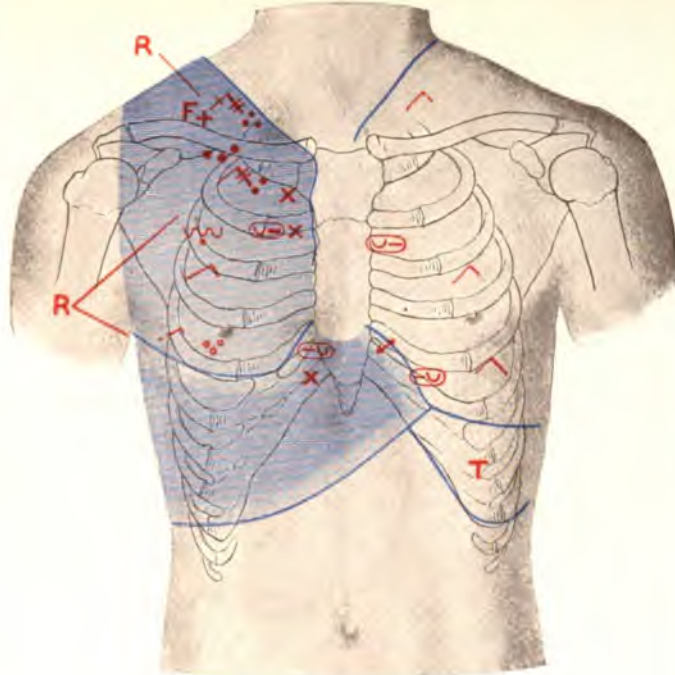
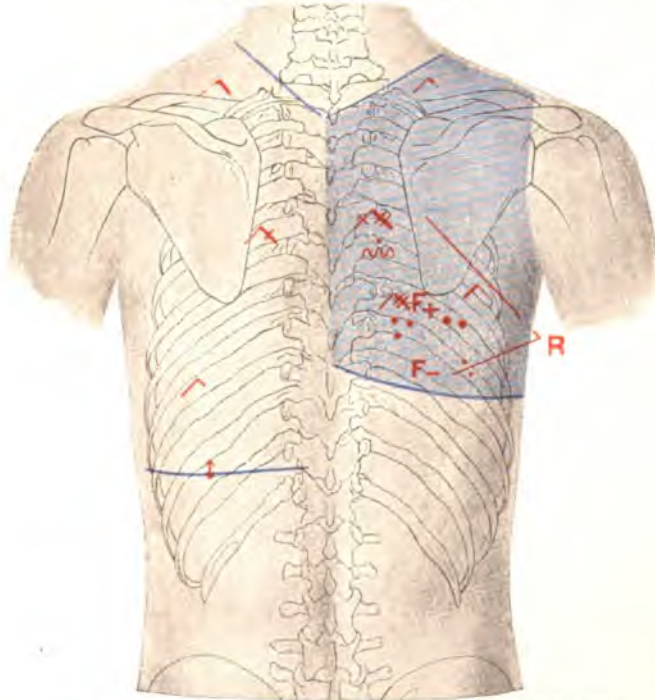


FIG. 2.—Posterior Aspect



Fibroid Phthisis with Chronic Pleurisy.

Heart drawn toward the right and aorta uncovered by retraction.



cirrhosis; it may be tuberculous in origin, or a manifestation of polyserositis (Concato's disease).

Symptoms.—Occasionally the patient may complain of a stitch in the side, but as a rule subjective symptoms are lacking.

Physical Signs.—In the milder degrees of chronic adhesive pleuritis there may be no physical signs except a slight indefinite friction rub and a slight impairment of resonance. As the condition becomes more extensive, there is noted diminution in movement of the affected side, dullness on percussion, absent or weakened fremitus, and distant breath-sounds. In the severe cases, there is great deformity of the chest from contractures of the affected side and compensating emphysema of the opposite side. There is considerable spinal curvature, deformity of the shoulder, and ingrowing and overlapping of the ribs. The heart may be drawn to one side or the other. The percussion note is dull, fremitus is absent and the breath signs distant or absent.

Chronic Pleurisy with Effusion.—This results either from an acute attack of pleurisy in which the fluid remains unabsorbed, or from a series of attacks, or it may develop slowly, apparently as a primary condition.

Symptoms.—These may remain entirely latent, or slight dyspnea may be observed. There may be an evening rise of temperature and acceleration of the pulse. Chronic effusions are likely to become purulent in children. The physical signs are those of an acute effusion.

Hydrothorax.—This is an accumulation of serum in the pleural cavity as a result of transudation. It occurs in the course of diseases which produce anasarca, as failing organic heart disease (right side affected), chronic nephritis (both sides affected), and debilitating diseases (usually on both sides). *The general symptoms* belong to the primary disorder. *Dyspnea*, at times severe, is usually the only local symptom. The physical signs are those of acute pleurisy with effusion from which it is differentiated by the absence of fever and the general characteristics of the fluid (see page 447).

Hemothorax.—Blood in the pleural cavity results from trauma of the chest wall, rupture of an aneurism into the pleural cavity, or from pressure upon the thoracic veins. The symptoms are those of internal hemorrhage and the physical signs are those of pleurisy with effusion.

Pneumothorax.—An accumulation of air in the pleural cavity practically always accompanied or followed by an outpouring of serous or purulent fluid, constituting respectively hydropneumothorax or pyopneumothorax. Pneumothorax may originate (1) externally, by perforation of the chest wall and pleura; (2) internally by perforation of the lungs, bronchi or esophagus; (3) as a result of infection of pleural exudates by the gas bacillus—*B. aërogenes capsulatus*. The most frequent cause is tuberculous disease of the lung and next an empyema. The disease is unilateral, the left side being affected not quite as often as the right.

Symptoms.—The onset is usually sudden, attended by *acute pain in the chest* and *excessive dyspnea with great dread of impending suffocation*. The patient sinks into collapse from shock, but sudden death is rare. If the escape of air into the pleural cavity is gradual there is less pain and dyspnea.

Physical Signs.—(See Plate XVII.) The affected side is *distended*; fremitus is abolished; the percussion note is a *bell-like tympany*, except when the distention is excessive and the air under great pressure, when the note is proportionately duller and higher in pitch; *dulness* is usually noted at the bases, the result of the accompanying effused fluid, and is *readily movable* with change of position by the patient; the diaphragm is depressed, pushing the liver or spleen down, and the heart displaced, particularly in a left pneumothorax when the areas of cardiac dulness may entirely disappear. Upon auscultation the most characteristic signs are elicited. The breath-sounds are feeble or absent except over the root of the lung where they are bronchial. If the lung is not completely collapsed amphoric breathing may be heard. A valuable sign is the *coin test*, the *bruit d'airan* (Trousseau). A silver coin is placed upon the chest and struck with another while the auscultator applies the stethoscope opposite the point struck. The ringing coin sound is reproduced with great intensity. It is almost pathognomonic and the outlines of a cavity can be traced by it.

The *hippocratic succussion splash* can be distinctly heard, often some distance from the chest wall, by shaking the patient.

Metallic tinkle on coughing or deep respiration is frequently heard and rales of a peculiar metallic quality are often observed.

Differential Diagnosis.—Pneumothorax may be confounded with a large *tuberculous cavity*, but the displacement of viscera, the coin test, and the absence of fremitus are not found in tuberculosis with extensive cavity formation.

Emphysema can be differentiated from pneumothorax by the fact that it is always bilateral, by the marked prolongation of expiration, and by the slow onset of the disease. The skodaic resonance above a *pleural effusion* and the dulness sometimes elicited in pneumothorax may cause the two conditions to be confused with each other, but in the former condition the metallic tinkle, the coin test, and the succussion splash are all absent.

Pyopneumothorax subphrenicus is differentiated from true pneumothorax (1) by the history of previous intra-abdominal trouble, more particularly gastric ulcer; (2) by the presence of the general symptoms of suppuration; (3) by the absence of movable dulness; (4) by the more marked displacement of the viscera beneath the diaphragm; (5) by the fact that the lower border of the lung moves upon deep respiration.

A *diaphragmatic hernia* may simulate a pneumothorax, but the history of accident and the peristaltic sounds heard by auscultation of the chest usually suffice to differentiate the two conditions.

PLATE XVII

FIG. 1.—Anterior Aspect.

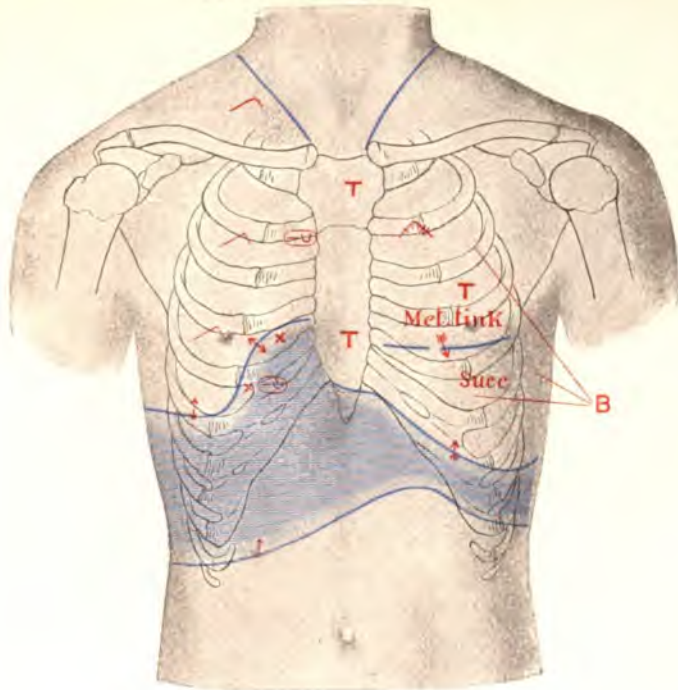
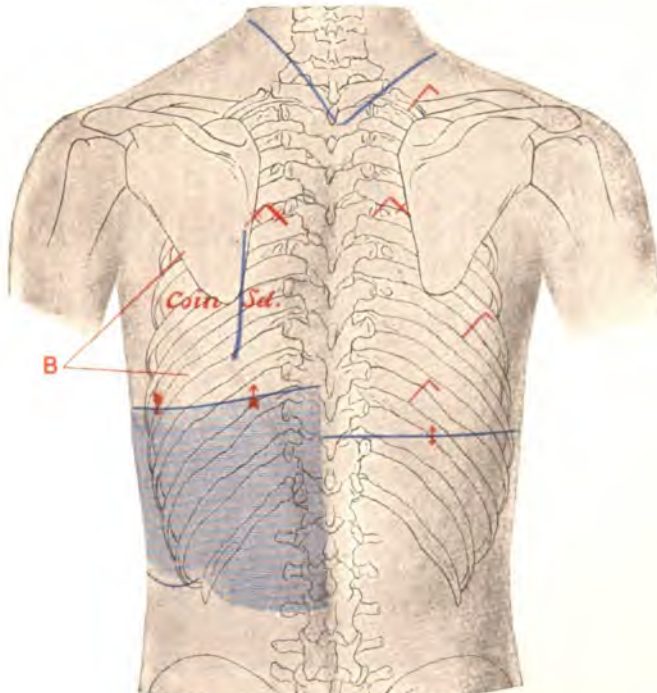


FIG. 2.—Posterior Aspect.



Pneumothorax (left-sided).



DISEASES OF THE MEDIASTINUM

Mediastinal Lymphadenitis.—Enlargements of the lymph glands of the mediastinum accompany either acute or chronic inflammatory affections of the lungs and bronchi. It is said that enlarged mediastinal glands can be demonstrated (1) by dulness on percussion in the interscapular region or when a difference cannot be elicited, by direct percussion over the upper dorsal spine; (2) by the presence of whispered pectoriloquy upon auscultating directly over the several spinous processes, below the first dorsal vertebra, where it is normally heard.

Suppurative Lymphadenitis.—This may result from ordinary inflammation, but is usually tuberculous in origin. The pus may perforate and discharge through the esophagus or a bronchus. The *x*-ray in either form of lymphadenitis will demonstrate the enlargement of the glands.

Mediastinal Abscess.—This rare condition may be acute, usually the result of trauma, less frequently secondary to erysipelas or the eruptive fevers; or chronic, generally tuberculous in origin. The anterior mediastinum is usually the seat of the involvement.

Symptoms.—Substernal throbbing pain, cough and dyspnea at times, with the general symptoms of sepsis are the most characteristic symptoms of an acute abscess. The abscess may point in the intercostal spaces, in the episternal notch or erode through the sternum.

Chronic Abscess.—This presents symptoms and signs similar to those of mediastinal growths. The *diagnosis* is based on the symptoms, the findings of a localized area of dulness anteriorly in the chest, the results of *x*-ray and the use of the exploratory needle.

Mediastinal Tumors.—Sarcoma and cancer are the most frequent forms of tumor in this region, the former usually primary, the latter secondary. Lymphoma, fibroma, dermoid, or hydatid cyst and gumma are occasionally found. The tumors may originate in the thymus gland, the lymphatic glands, the pleura, or the esophagus, most frequently in the thymus gland. The tumor, when one region alone is affected, is most frequently found in the anterior mediastinum. Males between thirty and forty are chiefly affected.

Symptoms.—The symptoms of mediastinal tumor are chiefly due to pressure. *Dyspnea* is early and constant, and may be laryngeal, or tracheal from pressure on respective parts of the respiratory tube. In some instances encroachment upon the heart or the vessels causes dyspnea. Again, the dyspnea may be due to a pleural effusion accompanying the growth. *Cough* of a peculiar character occurs. It is laryngeal and of a dry, brassy quality. Aphonia may arise from pressure upon the recurrent laryngeal nerves. (See Diseases of the Larynx.) If the bloodvessels are pressed upon, symptoms of obstruction occur, depending upon the vessel occluded. Edema of the upper extremities may occur. If the esophagus is pressed upon, there is difficulty in

CHAPTER XXXVIII

DISEASES OF THE HEART AND THE BLOODVESSELS

DISEASES OF THE HEART

General Considerations.—The symptoms of disease of the heart to the morbid process and to its physiological offices, are due to the anatomical structure of the organ. The heart is a hollow muscular structure which hangs in a cavity, the pericardial sac, and encloses other cavities—the two auricles and two ventricles—separated from each other by valves. Both sets of cavities are lined by serous membrane. The serous membranes are subject to similar lesions, and presents similar signs as diseased serous membranes elsewhere. In inflammation of the external membrane—the pericardium—the surfaces rub together and create a sound of friction. The external serous cavity may also become filled with the products of exudation or transudation, and as a result physical signs are produced. They are the physical signs of localized increase of contents as determined by inspection, palpation, and percussion, and of physical interference with the heart's action.

The serous membrane that lines the cavities of the heart and, with the subserous tissues, makes up the structure of the valves is subject to inflammations, the signs of which are common to all serous inflammations. The swellings and outgrowths that attend such inflammation occlude the orifices and prevent closing of the valves. There is produced a physical interference with the heart's function, which is recognizable by physical signs. The successful effort of the heart muscle to overcome such defects on the one hand (hypertrophy), or its failure on the other (dilatation), again leads to the production of symptoms and signs. The serous membranes, and hence the valves, are exposed to causes which excite inflammation. By virtue of the position of the heart at the centre of the circulation, the blood, containing at times certain endogenous and exogenous poisons or bacteria, constantly bathes the heart, and hence the organ is more likely to be affected by the deleterious material the blood carries than other parts. If the heart itself escapes, the toxins frequently spend themselves upon the arteries and cause degenerative arterial changes or directly irritate the vasomotor nerves, increase the blood pressure, and throw more work on the heart. The heart muscle is subject to the same morbid processes as other muscular structures. They are hypertrophy and atrophy; inflammation, acute and chronic, with overgrowth of connective tissue; and degenerations.

The heart is constantly subject to internal pressure. Dilatation of the cavities or of a portion of a cavity (aneurism) follows previous disease of the muscle or increase of internal pressure, and causes physical signs of enlargement. Degeneration of the heart muscle is also attended by symptoms of weakness and physical signs of enlargement (dilatation) or rarely of diminution in size (atrophy). When dilatation occurs, the orifices of the cavities enlarge, the valves cannot close them, and the symptoms and physical signs of incompetency and of blood regurgitation result. Hence the physical signs (objective symptoms) of cardiac disease may be due to primary and secondary morbid anatomical changes in the serous covering or musculature of the heart.

While the symptoms or signs of cardiac disease are often due to morbid processes in the heart or its membranes, it must be remembered that grave and persistent subjective and objective symptoms may be caused by, or at least associated with, disease of contiguous structures outside of the pericardium. The symptoms are not excited through the nervous system, but are produced by mechanical encroachment upon the organ, as in pleurisy with effusion, mediastinal disease, and disease of subdiaphragmatic viscera. They will be referred to in the study of objective symptoms. Care must be taken never to overlook the possibility of their presence.

In the study of the symptomatology of cardiac disease the student must bear in mind two things: (1) that the cause of the morbid processes and of the symptoms (*e. g.*, pain and palpitation) may be elsewhere than in the heart; and (2) that the ultimate object of the examination is to determine the muscular power of the heart. He will soon learn that with that power intact the functions can be performed, notwithstanding the presence of marked physical abnormalities.

The recognition of the condition or state of the functional efficiency of the heart depends largely upon the due appreciation of its physiological offices. Alterations in the physiological properties of the heart are recognized by the subjective symptoms of the patient and by the physical signs of hypertrophy and dilatation, changes in pulse-rate and blood-pressure, cyanosis, edema, etc. (See Functional Diagnosis.) The subjective symptoms, although due directly to alterations in the five inherent properties of the heart muscle (see Physical Diagnosis of Diseases of the Heart), for the most part arise from the secondary stasis of the blood in the peripheral circulation. The subjective symptoms are probably of greater value in determining mild or early cardiac insufficiency than the objective signs. They are (*a*) cardiovascular: pain, palpitation, arrhythmia, and pulsation; (*b*) pulmonary: dyspnea and cough; (*c*) gastro-intestinal: the symptoms of chronic gastritis and enteritis; (*e*) renal: symptoms of congestion of the kidney and of chronic inflammation; (*f*) hepatic: pain, fulness, and pulsation of the liver; (*g*) nervous: vertigo, headache, convulsions, coma, and disorder of the special senses.

The main subjective symptoms in diseases of the heart and blood-

vessels are common to many affections and are considered in the section on Subjective Diagnosis.

Pericarditis.—Inflammation of the Pericardium.—The inflammation may be acute or chronic. It is also divided according to the nature of the inflammation into simple fibrinous inflammation and inflammation with effusion. The effusion may be *serofibrinous*, usually the so-called second stage of the ordinary forms of simple fibrinous pericarditis; *bloody*, in tuberculous and cancerous pericarditis, and when in connection with hemorrhagic diseases; or *purulent*, due to tuberculosis, septicemia, or from extension of a purulent process from nearby structures. Pericarditis may be *primary* or *secondary*. The primary form is of extremely rare occurrence. Indeed, it may well be doubted whether pericarditis is ever primary or so-called idiopathic in origin.

Causes.—1. *Extension from Neighboring Structures.*—Extension of the inflammation from infected tissue in the vicinity is a common cause of pericarditis. It may follow a pleurisy and partake of the nature of the primary pleural inflammation. It often attends empyema either from extension of the infection to the pericardium or from rupture into the pericardial sac. It may follow all forms of inflammation of the mediastinum, and sometimes results from extension of infection from a tuberculous peribronchial gland. Disease of the ribs adjacent to the pericardium may set up pericarditis, acute or chronic. It attends aortic aneurism at times, but more frequently infectious endocarditis and myocarditis. Inflammations below the diaphragm frequently give rise to pericarditis: peritonitis, when general or local; subdiaphragmatic abscess; suppurative gastritis, with perforation of the stomach; abscess of the liver; suppurating hydatid cyst and other forms of suppuration below the diaphragm belong to these latter causes.

2. *General Conditions.*—Pericarditis arises in most cases in connection with the acute infectious diseases. Of these rheumatic fever is by far the commonest. Tuberculosis is also relatively common and septicemia, scarlet fever, measles, erysipelas, and typhoid fever may be the etiological factor at times; more rarely still the other acute infectious diseases. In the course of certain dyscrasias the pericardium is frequently the seat of inflammation because more vulnerable. This is particularly the case in scurvy. It occurs also in Bright's disease, and may be the first manifestation to the patient of this disease, particularly in the chronic form of nephritis. It occurs in the course of gout. In most chronic terminal illnesses a latent pericarditis occurs.

The various forms of pericarditis may occur at any age, although that which attends scarlatina and rheumatism occurs in early life, while late in life it is an attendant upon chronic Bright's disease and gout.

Acute Fibrinous or Plastic Pericarditis.—This is the most common form. It frequently attends Bright's disease, rheumatism, and tuberculosis. It may be wanting entirely in symptoms, although frequently

pain is present. The pain may be lancinating, dull or heavy; localized in the fourth or fifth interspace or referred like the pain in angina pectoris; and unmodified by pressure.

PHYSICAL DIAGNOSIS.—Inspection and percussion are usually negative.

Palpation.—A friction fremitus may be detected, due to the rubbing together of the roughened pericardial surfaces. It is most marked over the right ventricle, particularly in the fourth interspace, and is increased when the patient leans forward. The pulse rate is increased.

Auscultation.—A friction-sound is usually present. It may be present while the fremitus is absent; but, on the other hand, if the fremitus is present, we can always hear the friction. It is heard over the region where the fremitus is felt. (See Abnormal Sounds in Pericardium.)

DIAGNOSIS.—Acute pericarditis is overlooked because it is not sought for. In the larger number of cases there have been no indications of disease of the pericardium. The diagnosis is usually easy.

Differential Diagnosis.—The pericardial friction may be mistaken for an organic heart murmur or for pleural or pleuropericardial friction. (See Abnormal Sounds in the Pericardium.)

Pericarditis with Effusion.—As with fibrinous so with this form, it is frequently overlooked because it very often develops without symptoms. In plastic pericarditis we have referred to the occurrence of pain. This may occur before the effusion in the latter form, but it is usually moderate. As with dry pericarditis, however, it may, in rare instances, be very severe, anginoid in character, but is increased by pressure over the heart or in the pit of the stomach.

SYMPTOMS.—The symptoms are usually due to the special character of the inflammation and the presence of fluid in the pericardium.

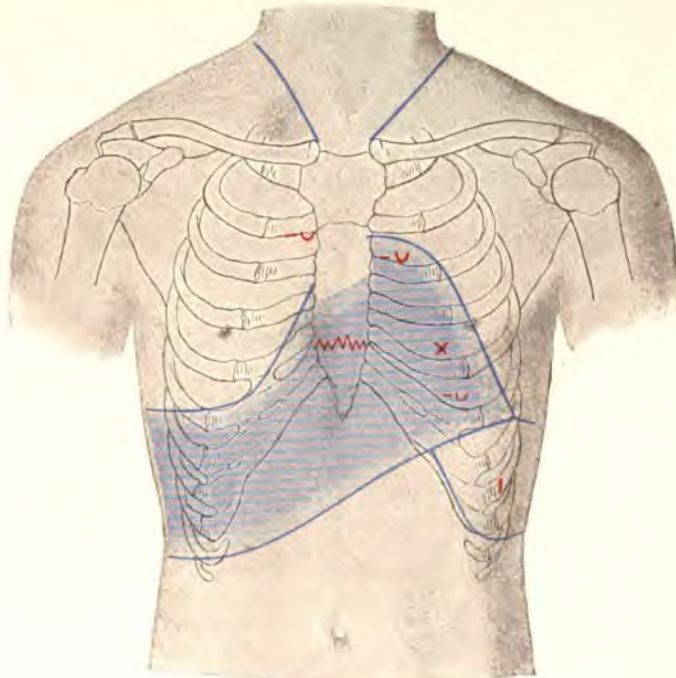
1. **General Symptoms.**—General symptoms are frequently latent but when manifest are usually cerebral. Delirium may be moderate or maniacal. It must not be confounded with the delirium which occurs in the course of acute rheumatism with hyperpyrexia. Other cerebral symptoms, as hemiplegia and convulsive attacks, occur in the course of pericarditis, probably due to an associated endocarditis, causing embolism.

The general symptoms of pericardial effusion depend upon the nature of the primary disease and the character of the fluid. In tuberculous pericarditis, emaciation, irregular fever, sweats, and prostration ensue. In purulent pericarditis there may be recurring chills with a temperature range decidedly intermitting, along with other phenomena of purulent accumulation.

2. **Local Symptoms.**—The local symptoms are due to the accumulation of fluid within the pericardium. Dyspnea is the most common. The degree depends upon the amount of effusion. If the latter is large, there may be extreme orthopnea; if the effusion is present for a considerable time, it may give rise to no symptoms. phonia, and

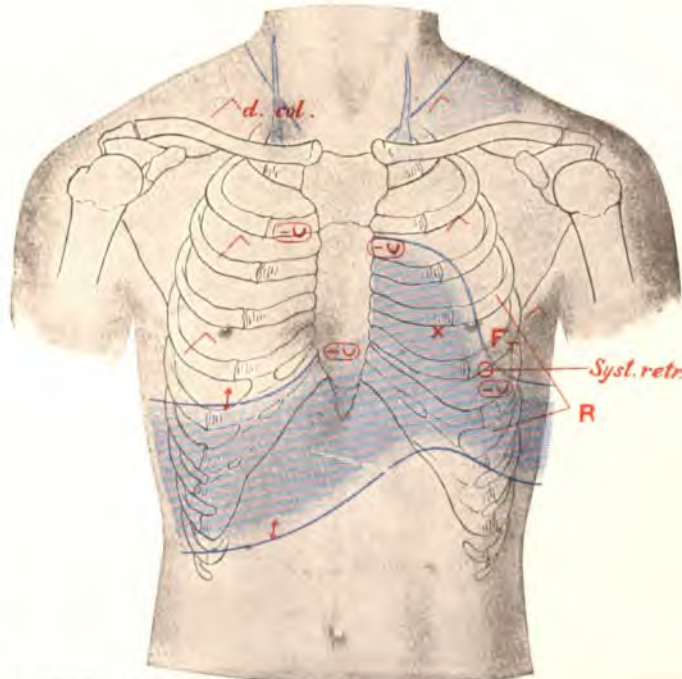
PLATE XVIII

FIG. 1



Pericarditis with Effusion.

FIG. 2



Adherent Pericarditis.

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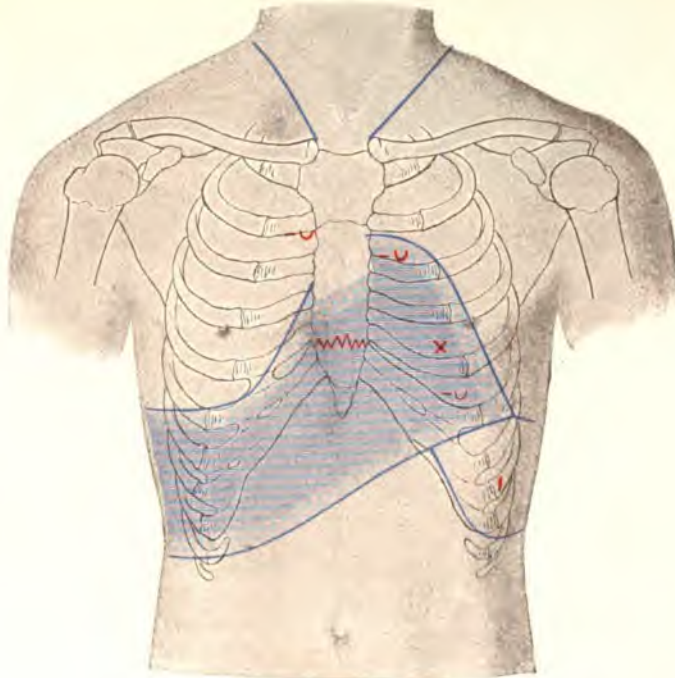
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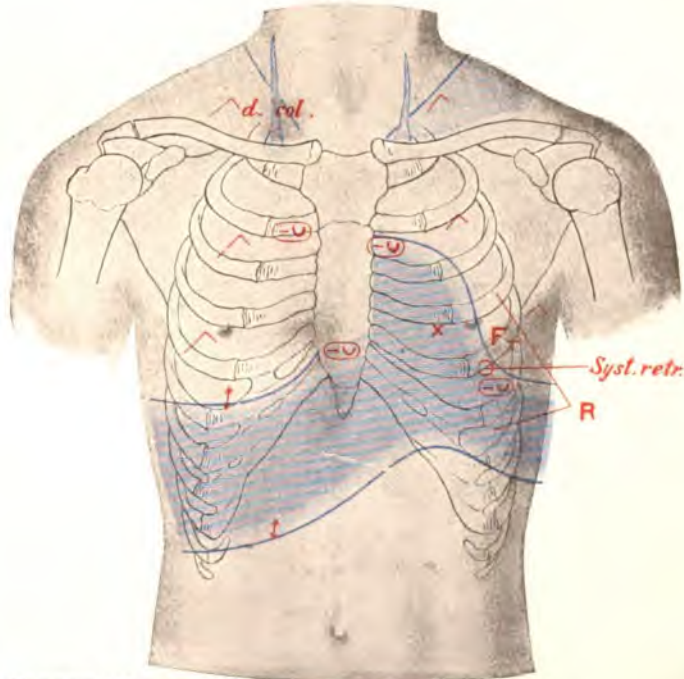
PLATE XVIII

FIG. 1



Pericarditis with Effusion.

FIG. 2



Adherent Pericardium. (Continued from page 17)



cough are frequent pressure symptoms. *Altered cardiac rhythm.*—The effect of the effusion upon the heart is to interfere with its action. Although usually regular, on the slightest exertion or the least excitement it palpitates violently or becomes irregular. The heart's action is increased in frequency; when the effusion is very large, it may be irregular. The *pulsus paradoxus* (fall of blood-pressure and decreased pulse frequency during inspiration) may be present.

PHYSICAL DIAGNOSIS (Plate XVIII, Fig. 1).—*Inspection.*—There is bulging of the precordia, particularly in children. The ribs and interspaces are prominent. In adults the interspaces are on a level with or distended beyond the surface of the ribs, and are sometimes widened. Expansion of the lung on the left side is diminished. The epigastrium may be prominent on account of displacement downward of the diaphragm and liver. The apex-beat is absent or faintly seen, displaced upward and to the left. It may be seen in the fourth interspace, or a faint impulse may be observed in the second and third interspaces beyond the midclavicular line.

Palpation.—The impulse is feeble and diminishes in force as the effusion increases. The position of the apex as determined by inspection is confirmed. The first rib is palpable at its sternal attachment in massive pericardial effusion. The liver in large effusions is depressed and readily palpable.

Percussion.—The area of precordial dulness is increased. There is increase of the lateral boundaries and great increase of absolute dulness, the relative dulness disappearing if the effusion is massive. The increase of the dulness if the effusion is not large gives a quadrilateral shape to the heart; if large, a triangular shape with the base down. The shape of the outline may vary with the position of the patient and flatness becomes more marked as the patient leans forward. The flatness may extend as high as the second rib. As pointed out by Rotch, dulness in the fifth right interspace in the angle formed by the right border of the heart and the right lobe of the liver is common in effusion. It is an early sign. Ebstein calls this region the cardio-hepatic triangle, and points out that the angle is obtuse, and that the dulness is absolute in effusion, although impaired in normal states because of proximity to the liver.

Pulmonary resonance is modified posteriorly. At the angle of the scapula is a small area of dulness, increased fremitus, and bronchovesicular and bronchial breathing when the patient is sitting upright, which disappears when he leans forward (Bamberger's sign). The dulness in large effusion includes the axillary region, so that it may simulate pneumonia or pleural effusion. The dulness, however, does not extend below the eighth rib in this region, whereas in pleural effusion dulness always extends to the bottom of the pleural sac. In a large pericardial effusion the semilunar space of Traube is obliterated.

Auscultation.—The heart-sounds are feeble, distant, but rapid. They may be scarcely heard at all over the precordial region. The

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sounds at the base of the heart are diminished in intensity. If a friction-sound was heard at the beginning, it disappears entirely as the effusion is poured out. In moderate effusions the friction may be heard when the erect posture is assumed.

DIAGNOSIS.—Pleural effusions may be excluded by the absence of dulness in the axillary region below the eighth rib; by increase in dulness beyond the right edge of the sternum; and at the same time by the absence of signs indicating dislocation of the heart to the right. It is said that pericardial effusion must be distinguished from dilatation of the heart. Although feeble and diffuse, the expansile shock of the impulse is more distinct than in dilatation, and an undulating wave over two or three interspaces can be seen. The area of dulness in dilatation does not extend upward except in cases in which the right auricle is enlarged. The dulness does not extend downward in dilatation without a similar displacement of the apex impulse. The shape of the dulness differs. In dilatation the dulness is square in shape; in effusion it is triangular or pear-shaped, with the base downward; furthermore, it changes with position. Normally, and in case of enlargement of the heart, the cardiohepatic angle is a right angle, sometimes even an acute angle; in pericardial effusion this angle becomes markedly obtuse. In dilatation the sounds are accentuated, and are of a valvular character; in effusion they are muffled. In pericardial effusion Bamberger's sign is of importance. A positive diagnosis can frequently be made by cardiocentesis. The x-ray is also of value.

The friction-sound may return after absorption of the effusion. It may disappear entirely and all signs of pericardial inflammation subside. Adhesion of the two layers of the pericardium may take place in plastic pericarditis and pericarditis with effusion.

Effusions into the pericardial sac of serum, blood, or air may take place without previous inflammation.

Hydropericardium.—This may occur in the case of general dropsy from kidney or heart disease. Rarely after scarlet fever, effusion into the pericardial sac may be the only dropsical symptom. The physical signs are those of effusion. It is not attended by fever. It is frequently overlooked, because investigation beyond the pleuræ is not made after an effusion into that cavity has been found.

Hemopericardium.—This occurs on account of rupture of an aneurism of the first part of the aorta, of the heart itself, or of the coronary arteries. Wounds of the pericardium and heart cause hemopericardium. Death usually takes place before there has been time to make an accurate examination. In the pericarditis of tuberculosis and cancer the effusion is frequently blood-stained. In asthenic and cachectic state and in the blood diseases (purpura, hemophilia, etc.) the same is true.

Pneumopericardium.—This occurs very rarely, and is due to perforation from without by a stab wound, or perforation from the lung, esophagus, or stomach. A purulent exudation may be due to *B. aërogenes capsulatus*, causing an accumulation of gas. If it arises from

perforation, acute pericarditis is set up. The accumulation of gas causes tympany over the movable area of percussion dulness. The most striking sign is noted on auscultation. Churning, splashing, or metallic sounds are heard, drowning the feeble heart-sounds. Death usually occurs quickly.

Adherent Pericardium (Plate XVIII, Fig. 2).—Chronic adhesive pericarditis may follow the acute form or, particularly if tuberculous, it may develop independently and progress slowly. It may be a mild form with simple adhesion of the peri- and epicardial layers or a giant form with chronic mediastinitis and adhesion of the pericardium to the pleura and chest wall. The condition may never be suspected nor recognized during life; it may be first suspected by the signs and symptoms of cardiac weakness or failing compensation; or it may be first noted on account of the appearance of marked ascites. The physical signs are necessary to make the diagnosis.

Inspection and Palpation.—Indrawing of the interspaces and at times the ribs may be seen at the time of the systole of the ventricles. This indrawing is most marked at the apex, and must not be confounded with the normal systolic retraction that occurs in the third and fourth interspaces. In some cases the systolic movement over the precordia is of an undulatory character. Broadbent calls attention to systolic retraction of the left back in the region of the eleventh or twelfth rib, a valuable sign. The apex is displaced outward and the area of impulse is increased, due to the attendant, always present hypertrophy. After the systole there is frequently felt a quick rebound, known as the diastolic shock, characteristic of pericardial adhesions.

In pericardial adhesions diastolic collapse of the cervical veins (Friedreich's sign), inspiratory swelling of the veins (Kussmaul), and the pulsus paradoxus are signs of but little value.

Percussion.—The area of cardiac dulness is increased usually upward, extending as high as the first interspace. The area of dulness is frequently not modified by respiration, that is, it is not lessened when the patient takes a full breath, when the lungs should expand over the precordial region. This is particularly the case when there is pleuritis associated with pericarditis, a common association in the large majority of cases.

Auscultation.—On auscultation the signs vary. The sounds are due to hypertrophy or to dilatation. In the former condition the first and second sounds are accentuated; in the latter, a murmur may be heard at the apex, loud and systolic in time.

In pericardial adhesions the physical signs depend upon the condition of the heart muscle at the time of the examination. At first we have the physical signs of hypertrophy, with retraction of the interspaces, particularly at the apex, or the space at the xiphoid cartilage. This is particularly the case in young subjects. In the later period of the disease the physical signs of dilatation arise, indicated by increase in transverse dulness, enfeeblement of impulse and of sounds, etc.

The physical signs of associated pleurisy aid in the recognition of adherent pericardium. Diminution of the breath-sounds, increase in the area of cardiac dulness, lessened fremitus in the neighborhood of the heart, pointing to pleural thickening, serve as further evidence. Sansom considers the presence of pulmonary tuberculosis of value, as pointing to the occurrence of pericardial adhesions, for the associated pleural adhesions are likely to be attended by tuberculous pericarditis.

Adhesive pericarditis is to be suspected in a young subject the victim of cardiac insufficiency when the symptoms do not yield to treatment, in short, when the heart is not affected by rest and digitalis.

In unusual cases the first evidence of adherent pericardium is ascites—a symptom-complex for which Pick has proposed the designation, “pericarditic pseudocirrhosis of the liver.” In reality the condition is a manifestation of multiple inflammation of the serous membrane, “multiple serositis,” or polyorhymenitis, of the Italians and others. The pericardium, pleuræ, and peritoneum (especially about the liver—*zuckergussleber*) may be involved consecutively.

Multiple serositis, or chronic adhesive pericarditis with ascites, is recognized by attention to the following facts: a history of a previous attack of acute pericarditis, pleuritis, or perihepatitis; the early occurrence and subsequent disappearance of the edema of the legs; marked ascites, with little or no edema of the legs; enlarged liver early in the course of the disease (in some cases the liver appears not to have been enlarged); small and distorted, but otherwise smooth liver in the later stages of the diseases; absent or very late enlargement of the spleen; repeated attacks of pain, tenderness, rigidity, and possibly palpable and audible friction in the right hypochondriac region—attributable to attacks of perihepatitis; rapid recurrence of the ascites after tapping, and the physical signs of adherent pericardium—without which, it may be said, the disease is incapable of diagnosis.

Diagnosis.—Briefly, fixation of the heart is the physical condition the signs of which are well summarized by A. O. J. Kelly as follows: in the diagnosis of adherent pericardium, most help will be derived from a weak or absent apex-beat, especially in cases in which there is no increase in the area of cardiac dulness; systolic retraction of a considerable area about the apex; systolic retraction of the base of the left chest posteriorly; arrest of the normal respiratory movements in the epigastric angle; imperfect descent of the apex-beat during inspiration; inadequate or entire absence of shifting of the apex-beat, with change in the posture (lateral posture) of the patient; absence of change in the limits of the cardiac dulness during the respiratory phase. absence of increase of the cardiac dulness to the right, despite marked engorgement of the veins of the neck; a diastolic shock or rebound at the heart; evidence of dilatation or hypertrophy of the heart in the absence of valvular or other disease that might cause it; absence of the characteristic changes in the heart in the presence of defini

valvular disease, that is, absence of the usual hypertrophy of the right ventricle in mitral disease and of the left ventricle in aortic disease; absence of pericardial effusion in the presence of pleural and peritoneal effusions; paradoxical pulse—inspiratory diminution in the force and volume of the pulse.

Endocarditis.—Endocarditis may be acute or chronic. In either form it is usually secondary. The acute form is divided into simple and so-called malignant, infectious or acute ulcerative endocarditis.

Simple Endocarditis.—Acute endocarditis rarely occurs primarily. It usually occurs secondarily to general morbid processes. The pathological antecedents are acute rheumatism, tonsillitis, whooping cough, scarlet fever, gonorrhea, rarely smallpox and typhoid fever. It is of common occurrence in pneumonia and tuberculosis. It is frequent in chorea. In the simple form it occurs in septic inflammations; debilitating diseases, as cancer, gout, Bright's disease, syphilis, predispose to it.

Symptoms.—The symptoms of simple endocarditis are scarcely observed during the early course of the disease. The process is latent and there are no indications of cardiac disease. The physical signs alone betray its presence. Unless these are sought for the disease is overlooked. The subjective symptoms are negative. In the course of rheumatism or chorea, or during convalescence from the former, the patient may complain of palpitation, and increased frequency and irregularity of the heart. At the same time there may be a rise in temperature, not attended by any increase of the rheumatic symptoms; this should direct attention to the possibility of a cardiac complication.

Physical Signs.—On examination a murmur is detected in one of the cardiac areas. The murmur is soft, low in pitch, and follows the laws of transmission according to its situation. Instead of a distinct murmur a roughening of the first sound alone may be heard. Preceding the murmur the heart's action may be quickened and arrhythmic; the first sound may change in character from day to day or be accentuated; the second sound may be reduplicated at the apex and accentuated. The new sounds may disappear at first when the patient sits up; later they persist. The murmur must not be mistaken for the murmur at the apex in cardiac dilatation, or for an extracardial murmur.

Malignant Endocarditis.—Unlike simple endocarditis, the malignant form very rarely develops in the course of rheumatism and chorea. (See the Infections.) It occurs more frequently in pneumonia than in any other disease. It arises in the course of erysipelas, septicemia, puerperal fever, and gonorrhea. It may occur in dysentery. It is usually a streptococcus infection, but may be due to the staphylococcus, pneumococcus, gonococcus, typhoid bacillus, etc.

Symptoms.—The general symptoms due to the specific morbid process are septic in nature. The febrile phenomena may be one of four groups: (1) The fever is paroxysmal. Chills and fever occur daily or at intervals of two or three days, resembling types of malarial fever. Each paroxysm is attended by profuse sweats. Rapid exhaustion

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ensues. The fever, instead of being distinctly intermittent, may be irregularly intermittent. (2) The fever is excessive and continued, and a typhoid state frequently sets in. The temperature is irregular; extreme prostration, low delirium, sordes, subsultus, and other symptoms of that condition arise. (3) The fever is moderate and continued. Physical examination, however, reveals the presence of marked endocarditis. In this group chronic heart disease has usually preceded the affection. The duration may be prolonged. (4) The fever may be remittent. Petechial rashes and erythema are common, so that, as pointed out by Osler, the disease may resemble the eruptive fevers. The sweating is profuse, contributing to the profound exhaustion which usually ensues. A septic diarrhea occurs. In a few rapidly fatal cases jaundice has occurred. Again, the symptoms may be almost exclusively cerebral, resembling cerebrospinal or basilar meningitis.

The embolic phenomena are due to escape into the blood-current of soft vegetations from the valves of the left heart (for the right heart is rarely affected), which are carried by the blood-stream to distant points of the circulation. Emboli occur in the brain, producing convulsions, aphasia, or hemiplegia; they occur in the retina, causing some visual defect which may be accurately recognized by ophthalmoscopic examination. They occur in the lungs, producing local pain, hemoptysis, possibly localized dulness, bronchial breathing, and rales. Emboli occur in the kidneys, producing bloody urine and renal pain. In nearly all cases the spleen is the seat of embolism, and in some instances infarctions may take place in this organ alone. The spleen is always enlarged, and the infarct may cause pain and increased tenderness on pressure. Emboli in the skin and mucous membranes present the most striking phenomena. The hemorrhages underneath the skin are minute. Emboli are seen in the extremities, but may also be found on the trunk. Occurring in the long bones, they cause local pain and tenderness. They occur in the mucous membranes, as those of the mouth and tongue.

Physical Signs.—Repeated examinations are necessary in some cases to determine the presence of a murmur, or to decide whether a previously existing organic lesion is the seat of an acute process. Variations in the character of the murmur from day to day are characteristic of malignant endocarditis. In organic heart disease with dilatation and failure of compensation, irregular fever followed by embolic phenomena points to the occurrence of an infectious process on the antecedent valvulitis.

Diagnosis.—The diagnosis rests upon proof that an infection is present. The history of an infection in some part of the body is most important in the diagnosis. When embolic phenomena are present, the diagnosis is made without much difficulty. A neutrophilic leukocytosis is present. The presence of the infection, as well as its nature, may be disclosed by blood culture.

The more pronounced general symptoms distinguish it from simple

endocarditis. The temperature range, the septic and typhoid symptoms, belong to the malignant form.

The more prolonged cases with moderately continuous fever, without apparent primary cause, are frequently confounded with typhoid fever. In endocarditis the onset may be more abrupt, and prostration and anemia may develop very early, and sweating may be profuse. The fever does not present the regularity of type that is seen in the development of typhoid. In endocarditis there is more chest oppression and dyspnea early in the disease than in typhoid fever. In endocarditis the course of the infection may be discovered in the genito-urinary organs, the lungs, the bones, etc. A polynuclear leukocytosis is practically always present in malignant endocarditis, while leukopenia is characteristic of typhoid. The results of bacteriological examination of the blood, urine, and feces, and especially the serum diagnosis, distinguish the two affections.

Malignant endocarditis must be distinguished from cerebrospinal fever and from smallpox of the hemorrhagic type. We must rely on the local cardiac symptoms and physical signs, and the preponderance of these over the other symptoms. Of course, the prevalence of an epidemic and a history of exposure are of service in the distinction between the diseases. Examination of the blood excludes the forms of malaria which formerly were mistaken for endocarditis.

There is probably no disease which is more frequently overlooked. It has been mistaken for cancer of the stomach with secondary infection, tuberculosis, pernicious anemia, Bright's disease with terminal infection, as well as the infections just considered.

Probably the local infection, which is causal, most frequently not sought for, is a latent gonococcus infection.

Chronic Endocarditis.—In some cases the acute condition seems to subside and then recur from time to time with the presence of the symptoms of the acute attack. In other cases the symptoms are less severe, but continuous for a long time, while in others persistent fever is associated with occasional attacks of chills and sweats. Such cases as these, with persistent symptoms lasting for months, usually terminate fatally in from one to three years.

Chronic Valvular Disease.—Chronic valvulitis may follow the acute form, and about 70 per cent. of all cases do so, or may develop in the course of arteriosclerosis due to alcoholism, syphilis, gout, etc. If associated with arteriosclerosis, the endocardial change may be part of the general degenerative changes which occur in that process. It may be of dynamic origin, often following prolonged heavy muscular exertion, by which the valves, particularly at the aortic orifice, have been subjected to repeated strain. The process is slow and insidious, and leads to the changes in the valve segments which constitute chronic valvular disease. Heredity seems to play a part in some cases. The mitral valve is involved in 59 per cent. of the cases of chronic valvular disease, the aortic in about 36 per cent., and the other two valves in

the remaining cases. The three most frequent lesions are mitral insufficiency, aortic insufficiency and mitral stenosis. Tricuspid insufficiency is frequent but practically always due to relative incompetency.

Chronic valvular disease includes valvulitis and valvular incompetency; there is either obstruction or regurgitation at the orifice affected. Valvulitis may exist with or without symptoms; valvular incompetency is always accompanied by symptoms. Valvulitis implies organic disease of the valves; valvular incompetency indicates regurgitation through orifices whose valves, whether healthy or diseased, are unable to effect perfect closure.

Valvulitis may be recognized by physical signs of (1) the lesion (2) the secondary effects of the lesion on the heart and circulation—hypertrophy or dilatation.

Valvular incompetency occurs usually in dilatation, and may be secondary to valvulitis. It is recognized by both signs and symptoms. Valvular disease is without symptoms so long as the heart muscle enlarges sufficiently to keep in balance the impaired circulation; compensation is then said to be complete. When compensation is broken we have the subjective symptoms enumerated above, all in consequence of dilatation of the heart from muscular insufficiency. It may be said that valvulitis is of no significance so long as compensation is perfect.

Compensation.—If an obstruction is moderate, and the person remains in good health, the hypertrophy is sufficient to overcome the obstruction. In this manner the effect of the valve lesion is compensated. On the other hand, when blood is permitted to flow by regurgitation backward into the cavity, that is, in the opposite direction to its usual course, it meets a blood-current flowing to this cavity in the normal direction, and the result is overdistention, or overfilling, of the cavity. Dilatation ensues, and may persist. If the regurgitation takes place suddenly, the dilatation continues; if gradually, as in chronic endocarditis, the dilatation is attended with hypertrophy. Thus, when there is regurgitation from the left ventricle into the left auricle, on account of incompetency at the mitral orifice, the auricle becomes overdistended with blood, for it is filling with blood from the pulmonary veins at the same time. This overdistention can only be overcome by hypertrophy. When this is not sufficient, the blood is obstructed in the pulmonary circulation, with the consequences hereafter to be mentioned.

In the consideration of valvular disease it is best to take up the symptoms of each valve lesion, bearing in mind that two or more of the valves may be diseased at the same time, or that both obstruction or regurgitation may be present at the same time at the same valve orifice.

Aortic Regurgitation, Insufficiency or Incompetency.—This may exist for a long time without presenting any symptoms. It occurs more frequently in men than in women, and is more common in the later

periods of life, in association with arteriosclerosis, more particularly with atheroma of the heart. It may be due to congenital malformation, to acute endocarditis, or to a sudden strain or undue exertion causing rupture of the valves. More frequently than any other cause, at least in persons under forty, is syphilis which sets up a specific aortitis, involving the valves secondarily. Relative insufficiency or incompetency is of very rare occurrence. Insufficiency is frequently combined with obstruction.

The blood falls directly into the left ventricle during the diastole. There is, first, a relative diminution in the amount of blood in the artery; and, second, an increased amount of blood in the ventricle, because the regurgitated column of blood meets the blood from the auricle which is filling the chamber at the same time. Dilatation of the left ventricle ensues, and is followed by hypertrophy. Dilated hypertrophy thus arises. The heart becomes enormously enlarged. This is one of the conditions in which enormous cardiac enlargement takes place—so-called *cor bovinum*—and is attended by arteriosclerosis.

SYMPTOMS.—They may be entirely absent so long as perfect compensation exists. This is particularly the case when there is but little general arterial sclerosis. Coincident lesions of other valves tend to break the compensation. The earlier symptoms are those due to arterial anemia, particularly anemia of the brain. They are headache, dizziness, and flashes of light before the eyes. The patient has an anemic appearance, and soon begins to suffer from shortness of breath. This at first develops upon slight exertion. Throbbing in the arteries, palpitation, and oppression about the chest are complained of, readily excited by undue exertion. Pain is a common symptom. It may be in the region of the precordia, of a dull, aching character, and radiate to the neck and down the arms, particularly on the left side. The anginoid pains may be followed by attacks of true angina pectoris. The latter are more common in aortic regurgitation than in any other valve lesion.

As compensation fails, venous stasis occurs and the dyspnea increases. The latter is worse at night, often paroxysmal, and compels the patient to sleep in a semi-erect posture. Congestion of the lungs takes place, giving rise to cough; hemoptysis occurs, but not so frequently as in mitral disease. Edema of the feet sets in, but general anasarca is not common. Edema of the feet may be due to the attendant anemia.

In aortic insufficiency sudden death is of common occurrence. This may take place at night during an attack of dyspnea, or occur suddenly upon the slightest exertion, such as straining at stool, or ascending a height, or walking more quickly than usual.

PHYSICAL SIGNS (Plate XIX, Fig. 1).—*Inspection.*—The apex-beat is downward, outward, and to the left. It may be as low as the seventh interspace, and as far as the anterior axillary line. The area of cardiac impulse is increased. It occupies the whole precordia, and heaving of the lower half of the chest may be seen. In young subjects there is precordial bulging.

SPERMAL REGURGITATION

Pulsation.—The impulse is strong and heaving. After compensation fails it is indefinite and vary. A thrill, diastolic in time, may be felt if the hand is placed above the middle of the sternum.

Pericardium.—The area of dulness is increased. The extent is greater than that in any other valve lesion, and the enlargement is more particularly downward and to the left.

Auscultation.—At the second costal cartilage on the right a murmur is heard, diastolic in time. This may be its seat of maximum intensity. It is transmitted along the course of the sternum toward the apex. In some instances the seat of maximum intensity is at the fourth left costal cartilage, or even at the apex. The second aortic sound is absent in the large majority of cases. However, both murmur and second sound may be heard at the same time.

Associate Murmurs.—Other murmurs also may be associated with aortic regurgitation, but not always due to disease of the aortic valve:

1. A systolic murmur at the second costal cartilage on the right, transmitted into the vessels of the neck, short, rough, and high in pitch. It is due to roughening of the valve segments, to atheroma of the aorta, to aneurismal dilatation of the aorta, or associated aortic stenosis.

2. *Flint's Murmur.*—A murmur at the apex, rumbling in character, localized to this area, usually presystolic in time. It is the murmur described by Flint, who attributed it to flapping of the mitral segments, which during systole are not forced back against the heart wall, on account of the dilatation of the ventricle. They remain in the blood-current and produce relative narrowing.

3. A systolic murmur in the mitral area, low in pitch, due to dilatation of the ventricle and consecutive incompetency of the mitral valves. This occurs when failure in compensation takes place.

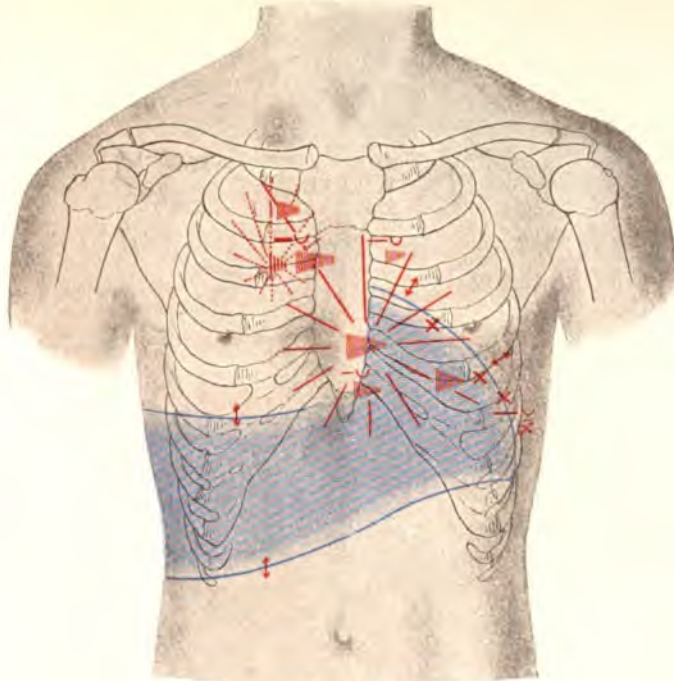
Examination of the Arteries.—Pulsation of the peripheral vessels is more common in aortic regurgitation than in any other valve lesion. The carotids throb, the temporals pulsate, the brachial and radial arteries are conspicuous. Pulsation of the retinal arteries is seen with the ophthalmoscope, and has often led to recognition of the disease. Pulsation of vessels in the fauces is occasionally seen. The pulsation is of a jerking character; in the neck it may simulate the pulsation of an aneurism. The aorta can be seen and felt at the suprasternal notch. The abdominal aorta pulsates vigorously in the epigastrium.

The pulse is significant in aortic regurgitation. The so-called 'water-hammer' or Corrigan's pulse is observed. The pulse is quick, of large volume, and jerking, and after striking the finger immediately recedes. It is most marked when the arm is extended vertically.

On auscultation of the large superficial arteries a loud so-called pistol-shot sound may be heard, due to the sudden filling of the unusually empty artery. Slight pressure with a stethoscope on the artery causes two murmurs, a normal systolic and a diastolic one, due to the regurgitation of the blood backward (Liesegang's sign).

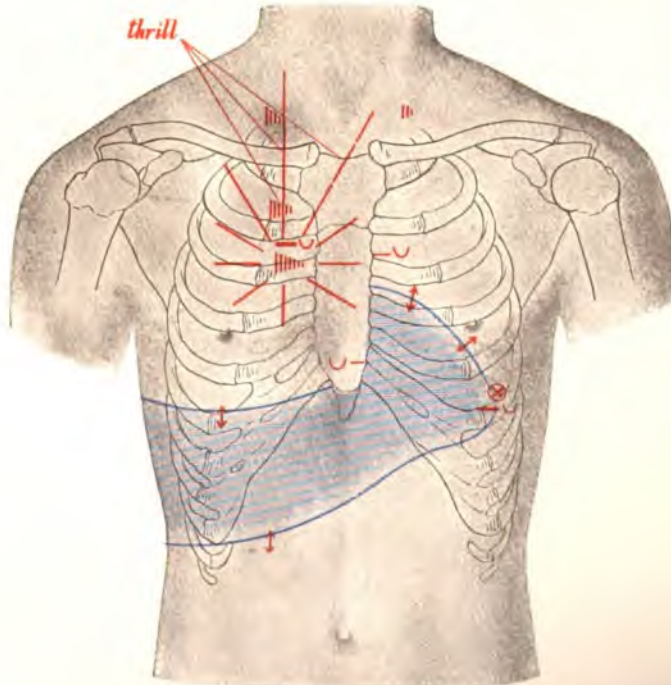
PLATE XIX

FIG. 1



Aortic Regurgitation.

FIG. 2



Aortic Obstruction.

may be musical. As the heart weakens, the intensity of the murmur lessens and its roughness disappears. It becomes soft and low in pitch. The second sound, if there is no regurgitation, is muffled or may be absent. The pulse is slow, small, and regular. The tension is usually increased.

DIAGNOSIS.—A systolic murmur at the aortic orifice may be due to aortic obstruction, atheroma or dilatation of the aorta, aneurism of the arch, syphilitic aortitis, or it may be accidental. Such a murmur is also common in aortic insufficiency without narrowing of the orifice and is due to stiffened valve leaflets projecting into the blood-current. Huchard describes a murmur in this situation, with vibratory thrill due to aberrant chordæ tendineæ. The murmur of aortic stenosis is distinguished from the others by its character, by the presence of thrill, by the character of the pulse, and by its association with hypertrophy of the left ventricle. A murmur due to atheroma of the aorta, particularly in the course of renal disease, is also associated with hypertrophy of the left ventricle. The diagnosis from aortic obstruction is often difficult or impossible. Slowness of the pulse is more characteristic of aortic obstruction. The accidental murmur is softer and low in pitch. There is no thrill, and the left ventricle is not hypertrophied. Accidental murmurs may be heard elsewhere. In atheroma the second sound is usually accentuated, and in anemia also it is intensified.

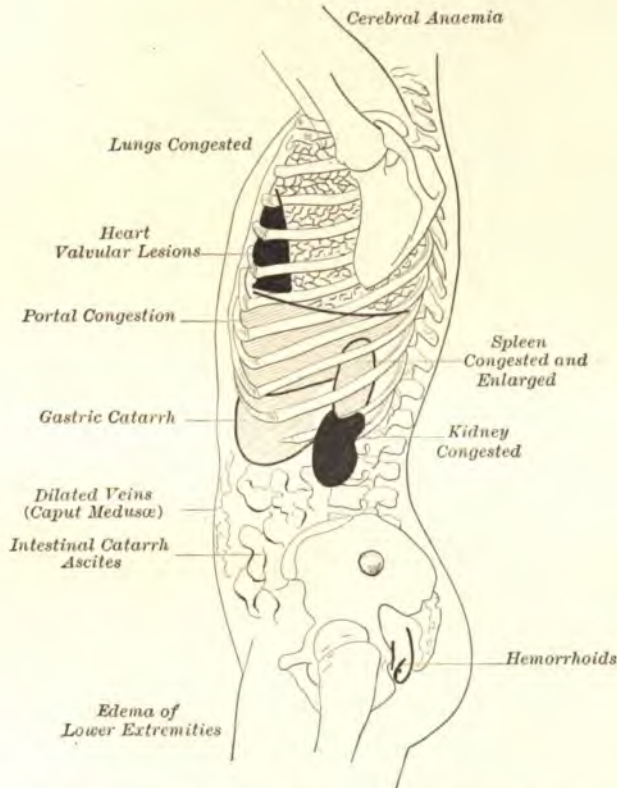
Mitral Regurgitation.—The regurgitation may be due to disease of the valves (organic) from previous endocarditis; or to inability of the segments to close the orifice (incompetence), enlarged from dilatation. Incompetence occurs in dilatation of the left ventricle, when the muscle is insufficient. The murmur of mitral insufficiency is one of the most commonly observed of all valve murmurs. Its ready production and often equally ready removal with treatment make it the least serious.

Mitral insufficiency or regurgitation has more serious effect upon the pulmonic and arterial circulation than any other valvular disease. The effects are as follows:

1. Dilatation and then hypertrophy of the left auricle occurs as a result of the back flow of blood.
2. As a result of the hypertrophy, a larger amount of blood is forced from the left auricle into the left ventricle; in order to remove the increased amount of fluid after the preliminary dilatation subsequent hypertrophy of this chamber follows.
3. On account of the overdistended auricle the pulmonary veins are not fully emptied during the diastole of that chamber. In consequence of the impeded flow of blood the vessels in the pulmonary circ are dilated and overdistended. The right ventricle is compelled act more vigorously, and even then cannot empty itself freely. Dilatation and hypertrophy of the right ventricle ensue.
4. This condition causes obstruction to the flow of the blood from the right auricle to the right ventricle: dilatation and hypertrophy of the right auricle follow.

If perfect compensation ensues through hypertrophy of both ventricles, engorgement in the lungs may not be observed. Moreover, the left ventricle is allowed to send out sufficient blood to supply the wants of the system. This compensation may continue for years. If it fails, from weakening of the muscle, a normal amount of blood is not distributed throughout the aortic area, but is thrown back upon (1) the left auricle; (2) the pulmonary circulation; (3) the right heart; and (4) the systemic veins. For a time the pulmonary circuit alone

FIG. 185



Showing the congestions following valvulitis with failure of compensation

is engorged, subsequently the systemic veins become congested because of dilatation of the right auricle and incompetence of the tricuspid valves. We then have the secondary effects of stasis upon the various organs of the body, with cyanotic induration and the development of dropsies.

Mitral incompetence without valvular disease is of frequent occurrence and is a condition which always attends hypertrophy and dilatation, which may take place from various causes.

SYMPTOMS.—As to the general symptoms, in a large number of cases perfect compensation may continue for a long time; no subjective symptoms arise, nor are there symptoms due to dilatation.

Compensation Moderate.—If compensation is not perfectly effected from the first, or is broken suddenly or gradually, the symptoms of dilatation arise. In patients in whom compensation remains only fairly good we have the characteristic appearance of heart disease. The face is pale and pinched, the lips and ears dusky, the capillaries of the cheeks enlarged, the finger nails clubbed, particularly in children; shortness of breath on exertion may be the only symptom complained of, and this may exist for years. The patients are, however, liable to attacks of bronchitis and of pulmonary hemorrhage.

Cardiac Symptoms.—Compensation Lost. Palpitation may occur in this as in other forms of heart disease, and from the same cause. When the compensation is broken, symptoms referable to the heart and to engorgement of systemic and pulmonary veins occur. Of the former, palpitation with a sense of oppression is the most common; pain is rare.

Venous engorgement leads to congestion, cyanosis, and dropsies. We now have the symptoms of dilated right heart superadded. The lungs are the first to be congested. Dyspnea becomes constant and is aggravated by exertion. Cough is present, excited by exertion or by speaking. With the cough there is bloody expectoration. Cyanosis occurs, and congestion of other organs follows. The liver is enlarged; obstruction in the portal area is prominent; chronic gastritis or gastro-intestinal catarrh ensues. The spleen is enlarged; ascites develops, and hemorrhoids and congestion in the rest of the portal area are seen. The kidneys are congested; the urine is scanty, albuminous, and contains casts and blood corpuscles. At the same time that the internal viscera are congested dropsies take place, beginning in the feet and extending to the rest of the body. Dropsy may have been present in the feet before symptoms of portal congestion ensued.

The patient may be relieved and compensation continue for a long time. Frequent attacks of dilatation of this character may take place, their recurrence being due to lack of care in hygienic matters, or failure of health from other causes. Finally, however, the compensation cannot be restored; it persists; the dropsies become more marked, and the symptoms of cyanotic induration and secondary sclerosis of the internal organs follow. It must not be forgotten that this is the chief form of organic heart disease seen in children.

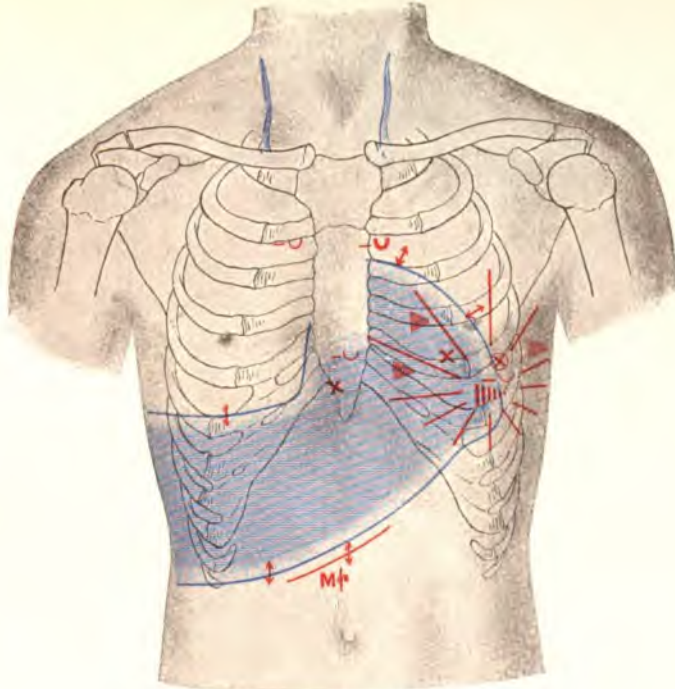
PHYSICAL SIGNS (Plate XX, Fig. 1).—*Inspection.*—On inspection the precordial area appears prominent; the apex-beat is displaced to the left and rarely downward. The cervical veins pulsate and are distended. The area of impulse is increased.

The Bloodvessels.—The amount of blood in the arteries is diminished. There is notable absence of visible pulsation in the arteries. The pulse at first is full and extrasystoles are common. It is notably



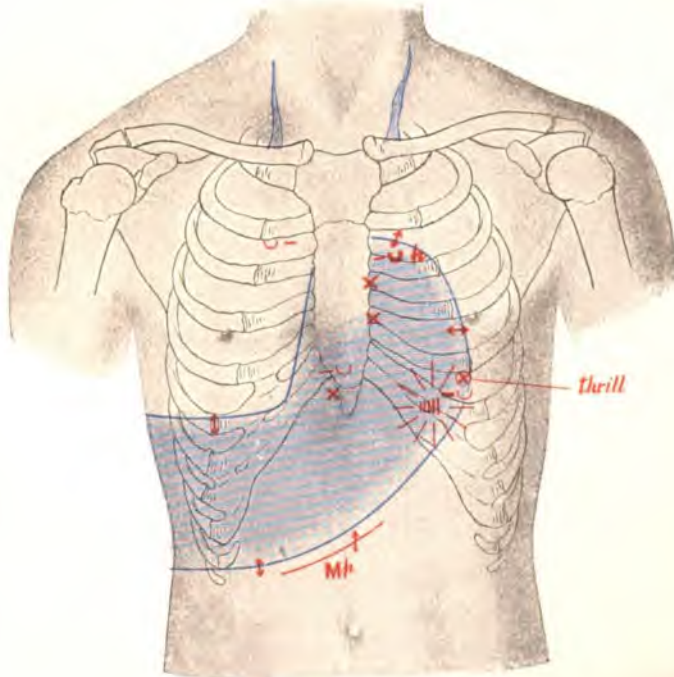
PLATE XX

FIG. 1



Mitral Regurgitation.

FIG. 2



Mitral Stenosis.

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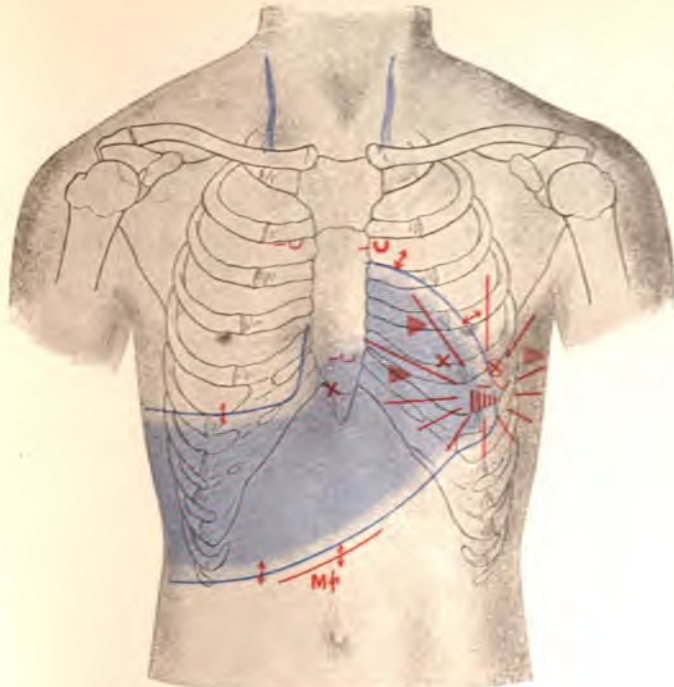
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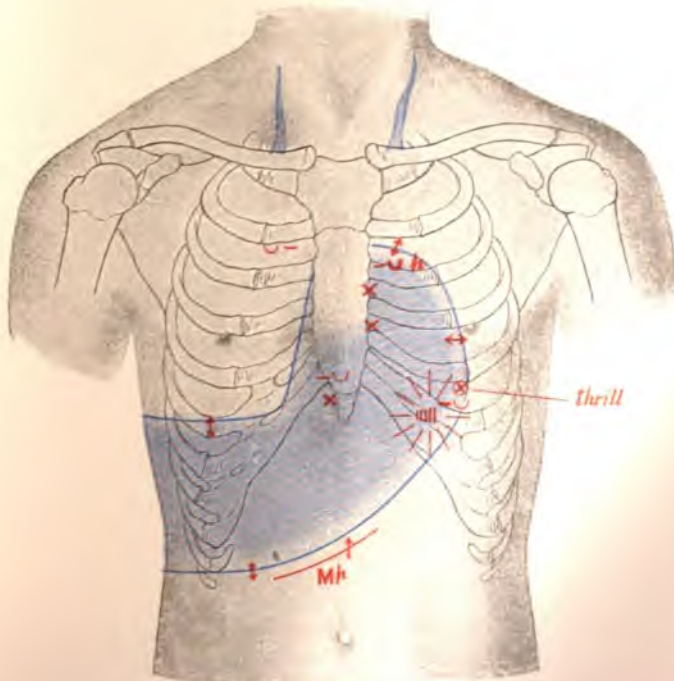
PLATE XX

FIG. 1



Mitral Regurgitation.

FIG. 2



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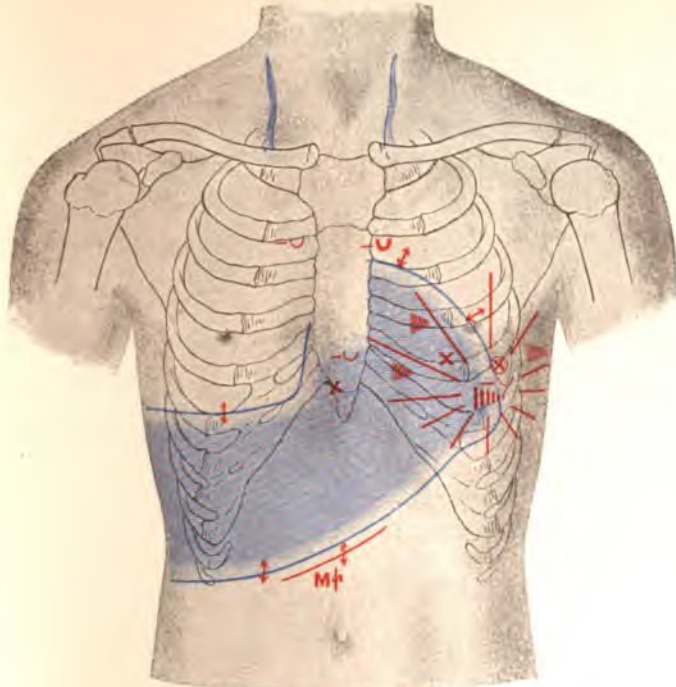
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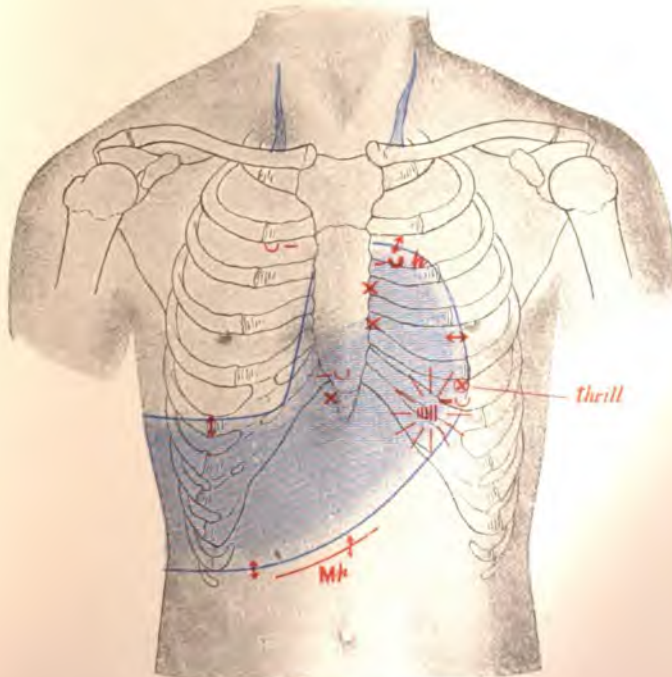
PLATE XX

FIG. 1



Mitral Regurgitation.

FIG. 2



Mitral Stenosis.

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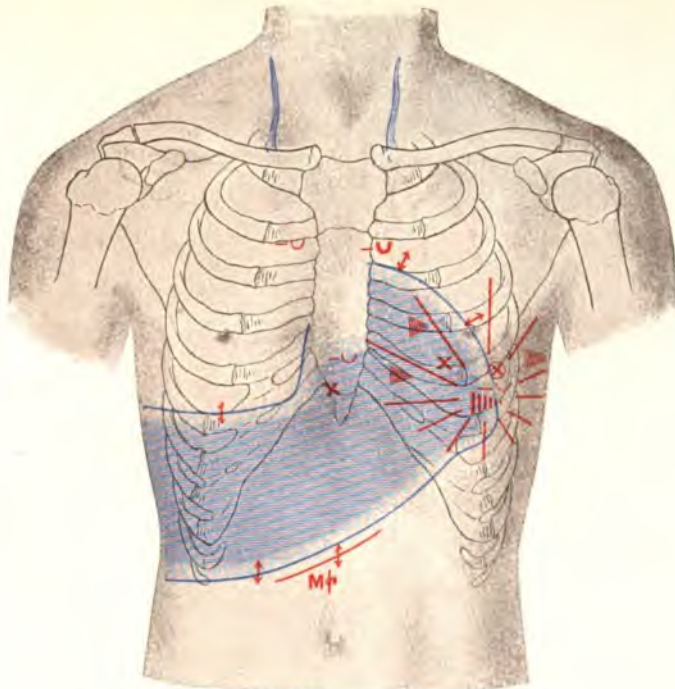
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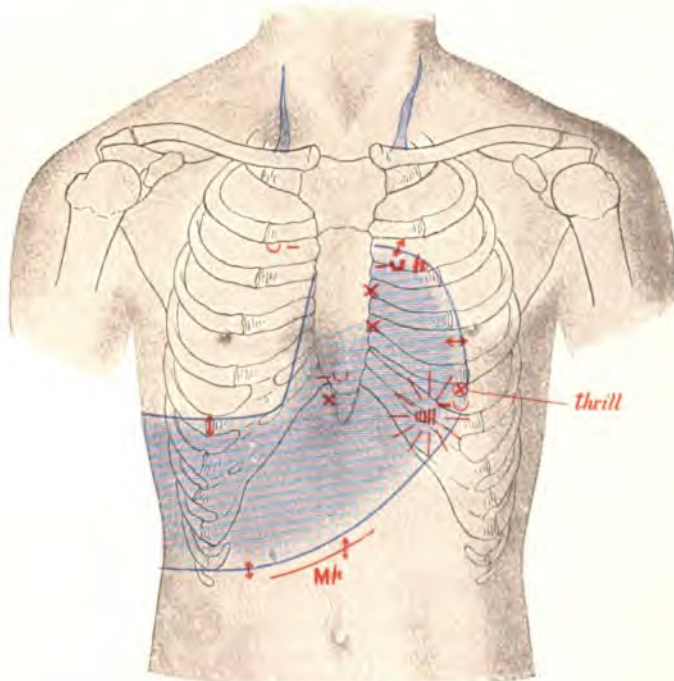
PLATE XX

FIG. 1



Mitral Regurgitation.

FIG. 2



Mitral Stenosis



small in volume and soft, usually becoming irregular with decompensation.

Percussion.—The area of dulness is increased transversely, but especially to the right. The area of dulness may extend beyond the right margin of the sternum to the extent of an inch or more and to the left as far as the midclavicular line, sometimes to the anterior axillary line.

Auscultation.—At the apex, the mitral area, a murmur is heard. The point of maximum intensity is in this region. It is systolic in time; and may replace the first sound entirely. It may be soft and low in pitch, or rough, high in pitch, even musical in character. It is transmitted to the axilla and the angle of the scapula. In some instances it may be heard loudest along the left border of the sternum. The pulmonary second sound is accentuated, most marked in the second left interspace. The murmur of mitral insufficiency is modified by the position of the patient and intensified after exertion. It may be present when the patient is lying down, and disappear in an erect posture. It may disappear when the patient is quiet and return after exertion. Other murmurs are sometimes heard: (1) a presystolic murmur, soft or rumbling, due to associated mitral stenosis; (2) when dilatation ensues, a low-pitched systolic murmur is heard at the ensiform cartilage and at the lower left border of the sternum. It is due to tricuspid regurgitation.

DIAGNOSIS.—Of special diagnostic significance are: the position of the murmur and the direction of the transmission; accentuation of the pulmonary second sound; enlargement of the transverse diameter of the heart, due to dilatation of both ventricles. The diagnosis is usually easy if the physical signs are sought for. It is important in the diagnosis to determine, if possible, the nature of the insufficiency, whether it is due to disease or incompetency of the valves. As previously mentioned, the history is possibly the only means by which a diagnosis can be made. If a mitral murmur ensues in old people in whom there has been physical cause for the development of dilatation and hypertrophy, as in emphysema or arteriosclerosis, it is usually due to relative incompetency of the valve. It must not be forgotten that the mitral area is the seat of a number of murmurs due to various causes.

Mitral Stenosis.—Obstruction to the flow of blood from the auricle to the ventricle is due to valvulitis. It is of much more frequent occurrence in women than aortic disease. It is much more often seen in young adults and children, because its etiological factors, rheumatism and chorea, are then more prevalent. Mackenzie says that mitral stenosis is one of the commonest forms of valvulitis, but it has not been recognized frequently on account of the concomitant regurgitation.

On account of the obstruction at the orifice changes ensue in the auricle. These changes depend in a measure upon the nature of the lesion. In the so-called button-hole contraction they are very marked.

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SYMPTOMS.—The symptoms were detailed in speaking of the mitral valve affections. They are those of obstruction in the pulmonary circulation and engorgement of the systemic veins.

PHYSICAL SIGNS.—*Inspection.*—The physical signs of dilatation of the right heart are seen. An impulse in the epigastrium is noted. This is seen especially between the xiphoid cartilage and the left margin of the ribs. Pulsation to the right of the sternum and in the second and third intercostal spaces may also be observed. The positive or ventricular jugular pulse may be seen in this condition and is always demonstrable by the polygraph. The pulsation is systolic in time. It is more marked in the right jugular than in the left. With the pulsation, regurgitation is readily observed by emptying the external jugular vein. Place the finger firmly on the vein just above the clavicle, and move it toward the head. The vein is thus emptied of blood; and with each systole of the heart it will be seen to fill from below in rhythmical pulsation. The veins are increased in size. This is more noticeable during the act of coughing or when the patient holds his breath in full inspiration. In rare instances the pulsation is transmitted to the subclavian and axillary veins.

Palpation.—By palpation the above conditions are also determined. The impulse over the lower sternum and in the epigastrium is noted to be forcible. The regurgitation pulsation is transmitted to the descending vena cava as well as to the ascending. The hepatic veins also distend during the systole. So-called pulsation of the liver is produced. With one hand on the fifth and sixth costal cartilages and the other over the liver in the axillary region, rhythmical expansible pulsation may be recognized. It is not of common occurrence, but is absolutely diagnostic of regurgitation at the tricuspid orifice. It must not be confounded with a pulsation transmitted to the liver from the heart or the aorta.

Percussion.—The area of cardiac dulness is increased transversely and upward, as described in mitral stenosis. It extends often far beyond the right edge of the sternum.

Auscultation.—A murmur is heard at the xiphoid cartilage, the lower end of the sternum, or the head of the fourth rib; it is systolic in time, usually low in pitch, and is heard clearly to the left of the sternum, within an inch of the apex, and to the right of the sternum and the outer limits of percussion dulness. It is not transmitted further. Other murmurs, if there is primary organic disease, are heard. If the heart is weak, the lesion may not be productive of a murmur. The pulmonary second sound is accentuated.

Tricuspid Stenosis.—Stenosis at this valve orifice is generally of congenital origin. In rare instances it may be secondary to lesions in the left heart. It is accompanied by dilatation of the right auricle.

Physical Signs.—The physical signs are the same as in stenosis at the mitral orifice, except for the alteration in their position. In some instances a presystolic thrill has been observed, and with it a presystolic murmur at the lower end of the sternum or toward the right of it. The

area of dulness is increased as in right-sided dilatation. Cyanosis is a prominent symptom and may be intense.

Diseases of the pulmonary valves are extremely rare and are almost always congenital.

Pulmonary Insufficiency.—The physical signs are due to regurgitation into the right ventricle. The maximum intensity is in the second left interspace, and the murmur is transmitted down the sternum. It cannot be distinguished from aortic regurgitation, except by the pulse.

Pulmonary Stenosis.—In stenosis of the pulmonary valve a systolic murmur and thrill are detected to the left of the sternum in the second interspace. The murmur is not transmitted to the vessels of the neck. The pulmonary second sound is weak. The effect on the heart is the production of right-sided hypertrophy.

Combined Valvular Lesions.—It must not be forgotten that there may be disease causing both obstruction and regurgitation at the same time and at the same orifice, or that two or more valves may be the seat of disease in the same individual. It is not impossible, for instance, to have aortic obstruction and regurgitation, mitral obstruction and regurgitation, and tricuspid regurgitation. Aortic obstruction or insufficiency is frequently combined with mitral insufficiency. Aortic and mitral insufficiency occur together most frequently in children; aortic obstruction and mitral obstruction in adults.

When more than one valve is diseased, the site of the various lesions is determined by the time, the position of maximum intensity, and the direction of transmission of the murmurs. Students often experience difficulty here. A systolic murmur may be heard in the aortic area and in the mitral area at the same time. If it is observed that each progressively weakens as the stethoscope is moved toward the middle of the precordial area, it may be inferred that the systolic murmur is due to two lesions. As previously intimated, the direction of the transmission of the murmur aids in the diagnosis.

Myocardial Insufficiency.—Under this broad head can be grouped the various conditions which cause symptoms referable to functional weakness of the heart muscle, usually with but frequently without demonstrable lesions of the myocardium. The demonstration of an actual myocardial lesion is of secondary importance. No matter what the disorder of the heart the chief diagnostic, therapeutic, and prognostic consideration are based upon the condition of the myocardium; upon what it can do and upon what is its functional efficiency. The following etiological classification is based largely upon conditions which may cause myocardial weakness. The conditions are all more or less associated and no sharp definite line can be drawn between them.

I. Congenital Weakness.—In a certain number of cases the heart seems to be physiologically inadequate and exhibits the signs of myocardial insufficiency without adequate cause.

II. Secondary Weakness without Myocardial Change.—The overabuse of alcohol, tea, coffee, tobacco, venery, etc., leads in a certain number of cases to myocardial weakness. Anemic and cachectic conditions may also cause myocardial weakness. If any of these primary conditions persist for any length of time they ultimately cause pathological myocardial changes which are discussed later. Excessive muscular effort may result in overstrain of the heart. Such a condition may develop suddenly, as in a boat race, or it may be the result of persistent severe muscular effort. Overstrain of the heart includes properly the conditions that develop as a result of continuous overwork of the heart as in valvular disease, pericardial disease, or in any of the conditions that cause hypertrophy without concomitant myocardial disease. Disease of the coronary arteries is so closely connected with myocardial disease (fibrosis) that it is more properly included in the degenerative myocardial processes.

III. Fatty Diseases of the Heart.—Excessive infiltration of fat into the myocardium or deposited around it practically always leads to myocardial insufficiency.

IV. Chronic Degenerative Changes in the Myocardium.—(1) Fibroid degeneration; (2) fatty degeneration; (3) amyloid disease. Probably the great majority of cases of myocardial degeneration arise as a result of sclerosis of the coronary arteries which is usually a part of general arteriosclerosis and is caused by the usual conditions that bring about sclerotic arterial changes (gout, chronic renal disease, plumbism, alcohol, syphilis, etc.). Any of the causes of myocardial insufficiency enumerated above may lead to myocardial degeneration. For example, chronic valvular diseases cause overstrain of the heart which ultimately results in disturbance of the myocardial nutrition (through the insufficient blood-supply) which in turn leads to fibrosis. Chronic myocardial degeneration may at times be a sequel of acute degenerative processes; it may be merely a manifestation of the aging process; or it may be caused by the extension of a peri- or endocardial lesion into the myocardium. In addition degenerative changes of the muscle may be the result of conditions which cause fatty degeneration (phosphorus poisoning, prolonged infection, wasting diseases, etc.) or amyloid degeneration (prolonged suppurating disease) of other organs.

V. Acute Degenerative Changes in the Myocardium.—(1) Acute interstitial myocarditis; (2) acute parenchymatous myocarditis. The acute degenerative changes arise as a result of the action of the toxins of acute infectious disorders, notably acute articular rheumatism, typhoid fever, diphtheria, pneumonia, pneumococcic and other pyogenic infections, typhus, smallpox, etc., particularly if the course of the disease is protracted. These changes may lead to fatty, hyaline, or amyloid degeneration and in favorable cases to fibrosis. At times extensive myocardial degenerative changes are found at autopsy without the occurrence of noteworthy symptoms during life. In such cases the myocardium is capable of fulfilling the demands upon

it during life. In other words, it was functionally efficient. Most myocardial degenerations, however, sooner or later reach a state in which the muscle is incapable of performing more than the ordinary demands upon it.

Symptoms.—The first manifestations of myocardial insufficiency are occasionally sudden in onset, more often of slow development. The cases that show symptoms sudden in onset usually develop the symptoms because of some severe muscular effort. At times the symptoms may appear suddenly as a result of some intercurrent disorder, as acute gout, an alcoholic debauch, or some such condition that puts a sudden strain on an insufficient heart. There will be palpitation, dyspnea, substernal distress, vertigo, and perhaps syncope. Examination of such patients will show marked precordial pulsation, increase of dulness to the right and a tricuspid systolic murmur with loud clear valvular sounds. The patient will be cyanotic, distressed and anxious-looking, and the pulse rapid and weak. Overwhelming symptoms may appear as a result of spontaneous rupture of a diseased heart wall (aneurism of the heart) or from a sudden shutting-off of the myocardial blood-supply as by thrombosis or embolism of the coronary artery.

The symptoms of myocardial insufficiency that develop slowly are more frequent than the acute conditions, yet often overlooked until advanced insufficiency of the muscle takes place. Frequently the myocardial condition is obscured by the primary disease, as in nephritis, emphysema, or arteriosclerosis, or it may be absolutely latent and without any clinical expression. The early manifestations of myocardial insufficiency are frequently referred to by the name "heart-tire." Dyspnea upon slight exertion, physical fatigue, dizziness, and digestive disturbances are first noted. Palpitation of the heart and precordial distress may be complained of. The heart is usually rapid, often irregular, and perhaps shows some increase in the area of dulness, after some slight exertion. The same symptoms may be first noted during convalescence from acute febrile disorders when the patient starts to get around, or the first symptom may be a sudden attack of heart failure which may terminate fatally.

In other cases the first manifestation of myocardial insufficiency may be paroxysms of angina. Still others may show the Stokes-Adams syndrome. Advanced cases of myocardial insufficiency show the phenomena of failing compensation and we find the patient continuously dyspneic, edematous, cyanotic, and complaining of symptoms that are referable to the passive congestion of the viscera.

Physical Diagnosis.—In the early cases and cases without myocardial degeneration abnormal physical signs of the heart are often wanting. In the more advanced cases myocardial changes usually always take place and the physical signs of the heart are those of (a) myocardial degeneration alone, or those of (b) myocardial degeneration plus some hypertrophy, or those of (c) myocardial degeneration plus dilatation.

The physical signs of myocardial degeneration are those of a quick, abrupt, forcible or absent impulse; or when palpable, of apex impulse displaced to the left; of marked increase in the area of absolute cardiac dullness and of characteristic auscultatory phenomena. The latter phenomena are those either of systolic shock, greater than the force of the impulse would lead us to believe to be present, or of feeble muscular sound. In the early stages the former exists, and continues later if hypertrophy coexists. From the first, or at least early, we have gallop rhythm, or reduplication of the systolic sounds. This reduplication may be heard over the right heart or more distinctly over the left heart; sometimes it is heard all over the precordia. It may be more marked in the supine position, and is generally more marked after exertion. It may disappear after a stimulant is taken or if the heart is stimulated by fever. Reduplication of the second sound also obtains, but is less frequent in the degeneration of coronary artery disease than in that due to valvulitis. In uncomplicated cases murmurs are not heard until late in the disease. Sometimes, however, we hear a systolic murmur at the fourth rib, greater in the recumbent posture. This murmur is soft, low in pitch, and so often heard in the left sternal line that it is my custom to call this line the myocardial line. However, it may be at the tricuspid or even in the pulmonary area—when it is probably, although not necessarily, accidental.

Arrhythmias are common, particularly extrasystoles, of auricular but more frequently of ventricular origin. Auricular fibrillation may also occur, usually in the later stages of the condition when dilatation has taken place. The blood-pressure is high and the artery shows signs of sclerosis in most cases.

When dilatation supervenes, the physical signs change in keeping with the physical condition of the heart. The auscultatory signs of insufficiency at the mitral and tricuspid orifices are predominant, rarely at the aortic; accentuation of the pulmonary second sound is a frequent sign, with engorgement of the venous side of the circulation.

Diagnosis.—The diagnosis of functional insufficiency of a low grade is difficult. (See Functional Diagnosis.) High-grade myocardial insufficiency is a condition usually readily recognized, although greater reliance should be placed on the age of the patient, the history and the symptoms, than on the physical findings, which are frequently misleading and often incorrectly interpreted.

Aneurism of the Heart.—Aneurism of the valves, following endocarditis, cannot be recognized during life. Aneurism of the walls usually results from a localized chronic myocarditis following infarct. The aneurism develops usually in the left ventricle at the apex. The symptoms are indefinite. In rare cases a marked bulging has been noted in the region of the apex, and the tumor may perforate the chest-wall. A projection beyond the normal line of cardiac dullness may be detected by stethoscopic or plessimetric percussion. The symptoms are those of myocardial insufficiency and of dilatation of the heart.

PLATE XXI

FIG. 1

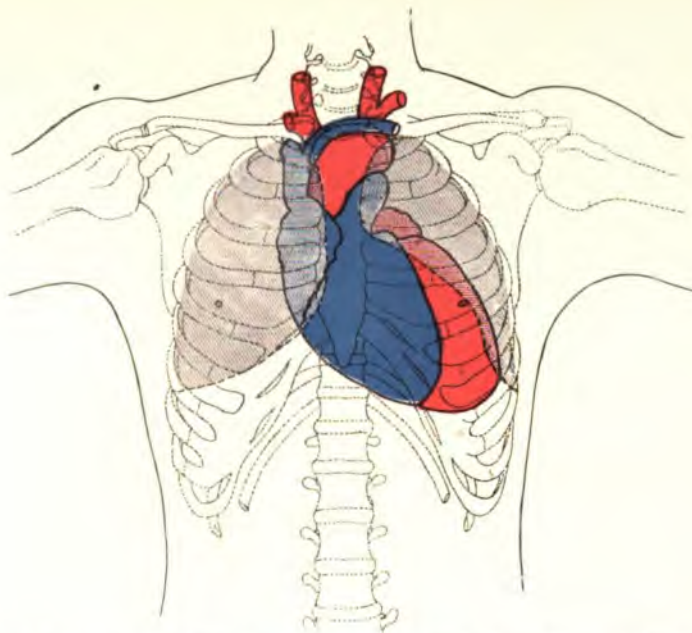


Diagram to illustrate the size and position of the heart and adjacent bloodvessels, the parts of the heart covered and uncovered by the lungs, and the superficial and the deep cardiac dulness in aortic insufficiency. RED—the left side of the heart (marked hypertrophy and dilatation). BLUE—the right side. The DARK area represents the part of the heart uncovered, the LIGHT area the part covered, by the lungs.

FIG. 2

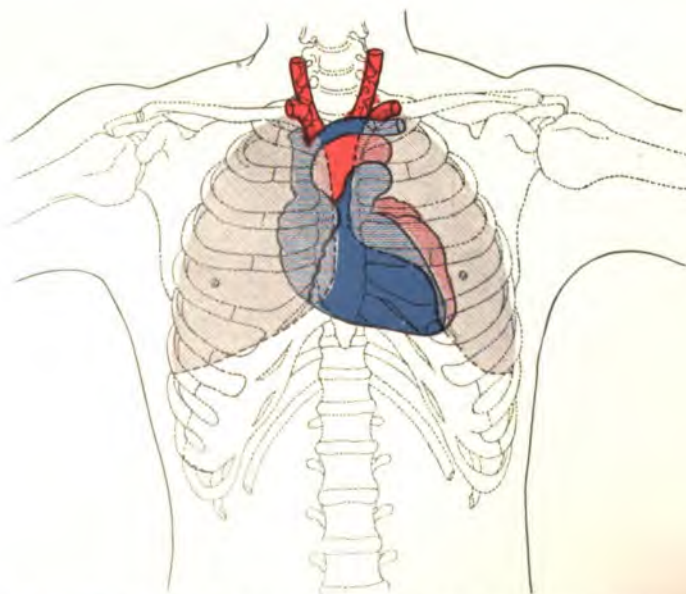


Diagram to illustrate the size and position of the normal heart and adjacent bloodvessels, the parts of the heart covered and uncovered by the lungs, and the superficial and the deep cardiac dulness. RED—the left side of the heart. BLUE—the right side. The DARK area represents the part uncovered by the lungs, the LIGHT area the part covered by the lungs.



PLATE XXII

FIG. 1

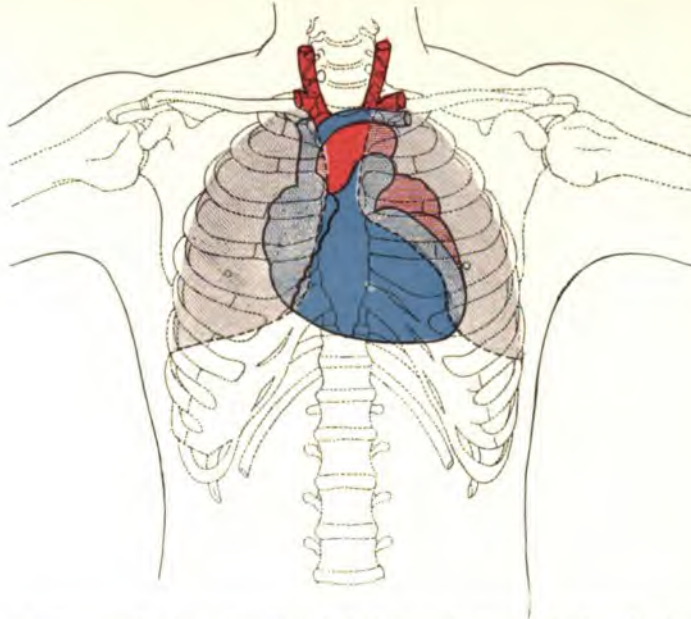


Diagram to illustrate the size and position of the heart and adjacent bloodvessels, the parts of the heart covered and uncovered by the lungs, and the superficial and the deep cardiac dulness in mitral stenosis. RED=the left side of the heart (hypertrophy and dilatation of the auricle). BLUE=the right side (hypertrophy). The DARK area represents the part of the heart uncovered, the LIGHT area the part covered, by the lungs.

FIG. 2

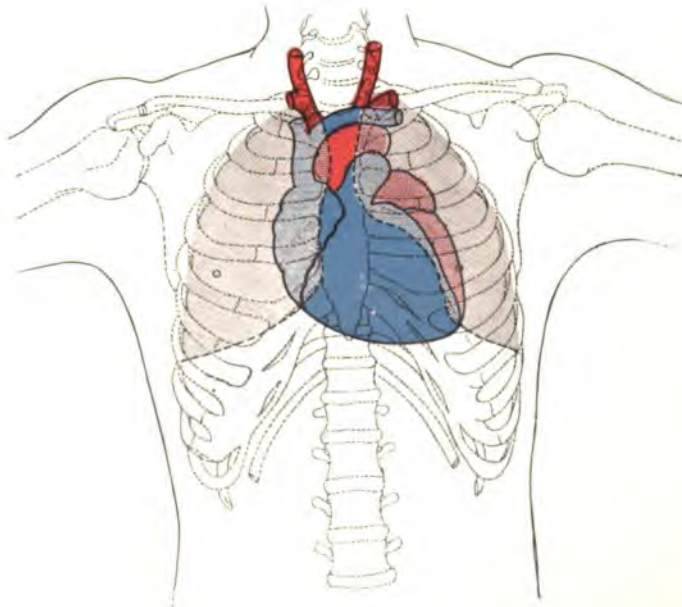


Diagram to illustrate the size and position of the heart and adjacent bloodvessels, the parts of the heart covered and uncovered by the lungs, and the superficial and the deep cardiac dulness in mitral insufficiency. RED=the left side of the heart (hypertrophy and dilatation). BLUE=the right side (hypertrophy). The DARK area represents the part of the heart uncovered, the LIGHT area the part covered, by the lungs.



Rupture of the heart is one of the causes of sudden death, often without previous symptoms. Aneurism of the heart, acute softening (myomalacia cordis) from coronary obstruction, suppurative myocarditis, and gumma are the usual antecedent conditions. The accident takes place during exertion. Quain collected 100 cases, in 71 of which death took place without warning. In other instances there was a sense of anguish and suffocation in the cardiac region. The physical signs of slowly developing pericardial effusion may be obtained if the leakage from rupture is slow in progress.

Tumors and Parasites of the Heart.—Benign tumors of the heart are exceedingly rare; carcinoma and sarcoma are somewhat more common, but are usually secondary, and the symptoms are obscured by the primary lesion unless decompensation occurs. Parasites are rare. The most common is the echinococcus, which usually involves the right ventricle.

Enlargement of the Heart.—Enlargement of the heart is due to dilatation. In hypertrophy there is increased thickness of the muscular walls. This may be general or limited to the walls of one chamber. Hypertrophy is further divided into simple hypertrophy, in which the cavity or cavities are of normal size, and eccentric hypertrophy, in which, with increase in the wall, there is enlargement of the cavities. This is hypertrophy with dilatation. The left ventricle is most frequently the seat of hypertrophy, when one chamber is involved. The cause of hypertrophy is increased work upon the heart which is followed by increased size of the muscle.

General Hypertrophy.—This occurs from overaction, from disorders of the heart itself, or from affections of the bloodvessels.

A. Disorders of the Heart.—(1) Diseases of the aortic valves; (2) pericardial adhesions; (3) myocarditis of the fibrous variety; (4) neuroses with overaction and frequent palpitation, as in exophthalmic goitre and from the effects of tea, tobacco, and alcohol.

B. Affections of the bloodvessels which cause hypertrophy are (1) general arterial sclerosis; (2) increased arterial tension as in Bright's disease, and in toxemias from lead, the poison of gout and of syphilis; (3) increased blood-pressure from prolonged muscular exertions; (4) narrowing of the aorta from external pressure and from congenital stenosis or the development of an aneurism.

HYPERTROPHY OF THE LEFT VENTRICLE.—Disease of the aortic valves, mitral valvulitis with regurgitation, and those diseases of the vessels which cause general hypertrophy also cause left ventricle hypertrophy.

HYPERTROPHY OF THE RIGHT VENTRICLE.—Obstruction to the flow of blood in the pulmonary area is the usual cause of hypertrophy of the right ventricle. This obstruction occurs in lesions of the mitral valve, causing increase in pressure of the pulmonary circulation; and in diseases of the lungs as in emphysema, cirrhosis or chronic bronchitis. It occurs if there is disease of the right heart with obstruction of the

without the grave local changes in the heart, or secondary changes in the peripheral vessels that occur in left ventricle hypertrophy. Pulmonary congestions and apoplexy may occur.

Physical Signs.—The physical signs of hypertrophy of the right ventricle have been referred to under the various valve affections. There is bulging of the lower part of the sternum and cartilages. An epigastric impulse in the angle between the ensiform cartilage and the ribs is noted. The impulse may be in the sixth interspace. It is diffuse; it may extend upward as in mitral stenosis. Cardiac dulness is increased toward the right, 2 cm. or more beyond the border of the sternum. The pulmonary second sound is accentuated and reduplication may take place. The radial pulse is small. If there is tricuspid regurgitation, the physical signs that attend it are present. Hypertrophy of the right auricle occurs under the same conditions.

HYPERTROPHY OF THE LEFT AURICLE.—This is present in mitral stenosis and the physical signs are those of increase of dulness to the left of the sternum in the second and third interspaces.

Diagnosis.—The forcible impulse in nervous palpitation of the heart must not be confounded with true hypertrophy, although it must not be forgotten that hypertrophy frequently follows neurotic palpitation, as in the smoker's heart, or in exophthalmic goitre. The enlargement must not be confounded with enlargement of the area of cardiac dulness in the precordial region from other causes, such as pericardial effusion, aneurism and mediastinal tumor, pushing the heart against the chest wall; disease of the lungs, on account of which they are withdrawn from the surface of the heart, as in phthisis or chronic pleurisy; and displacement of the heart from pressure, as in effusion on the left side of the chest or in disease below the diaphragm. The cause of hypertrophy should be ascertained, for it is a valuable aid in diagnosis. It must not be forgotten that emphysema of the lung may mask a considerable hypertrophy of the heart by causing diminution of the area of dulness.

Dilatation of the Heart.—Enlargement due to dilatation of the heart is common. The condition usually succeeds hypertrophy. Thickening of the muscles attends dilatation of the cavities, as in dilated or eccentric hypertrophy. The dilatation occurs because of increased pressure within the cavities or because of weakening of the heart walls, the pressure within being normal.

1. Increased pressure within the walls is due to an increased amount of blood within the chamber from regurgitation, or from an obstacle to the outward flow of blood. Simple hypertrophy occurs first in many cases; in others, hypertrophy with dilatation; in not a few, dilatation takes place at once. In dilatation the chamber does not empty itself during the systole. It is seen physiologically after the exertion of ascending a great height. It may remain within the bounds of physiological action. The dilatation may be temporary, as is demonstrated after running violently, by increased epigastric pulsation and increased

cardiac dulness. The tricuspid valves temporarily become incompetent, owing to their safety-valve action. The latter may continue after the acute strain, the heart always showing symptoms of the condition; or it may disappear entirely. An excessive dilatation results in heart-strain, with cardiac distress and dyspnea, symptoms due to over-distention and paralysis of the heart. Dilatation occurs in all forms of heart lesion previously described. The most typical form is seen in aortic regurgitation when the left ventricle is affected and in mitral regurgitation when the left auricle becomes the seat of dilatation.

2. Disease of the heart walls, lessening the resisting power, the normal pressure within the cavities being maintained, invites dilatation. Acute dilatation may ensue in acute myocarditis in the course of infections, as pneumonia, scarlatina, typhoid fever, rheumatic fever, and erysipelas. Certain changes which the heart muscle undergoes in acute endocarditis and pericarditis may lead to dilatation. In anemia and chlorosis the same process may take place. In chronic myocarditis dilatation takes place at the apex. When pericardial adhesions are present, the fibrinous overgrowth invades the interstices of the myocardium, thereby weakening the heart muscles. Dilatation may follow.

SYMPTOMS.—The symptoms of dilatation are the reverse of those of hypertrophy. When compensation fails, the blood is not expelled from the chambers in systole, so that the cavity is overdistended with blood which accumulates during the diastole. As soon as dilatation becomes permanent, incompetence of the valves takes place. In aortic obstruction the left side is first affected. It may be compensated for by hypertrophy of the right side. When this fails, venous engorgement and dropsy ensue. The symptoms have been described under Chronic Valvular Disease (Compensation, page 626). In acute dilatation there is a sudden occurrence of dyspnea, and pain, or at least precordial oppression may be complained of. The heart's action increases in frequency. The pulse is rapid, feeble, irregular, and may be barely palpable at the wrist.

PHYSICAL SIGNS.—*Inspection.*—The apex is displaced to the left, even as far as the axillary line, but rarely downward unless the hypertrophy precedes the dilatation. The impulse is diffused and undulatory in appearance. The maximal impulse is defined with extreme difficulty. It may be visible when the patient leans forward, yet not felt.

With the diffuse area of impulse a quick impulse may be felt—much weakened, however. When the right ventricle is dilated, the impulse is seen and felt to the right or left of the xiphoid cartilage, and there is a wavy pulsation along the left edge of the sternum in the fourth, fifth, and sixth interspaces. If the dilatation is extreme, involving the right auricle, a pulsation at the third right interspace close to the sternum may be felt. Tricuspid regurgitation is then present.

The area of dulness is increased in the same directions as in hypertrophy, if the two coexist. In general, it may be said that the increase extends outward to the right or left, the direction corresponding to

the ventricle affected. When the whole heart is dilated, the increase of dullness is in a transverse direction on both sides. The apex is round or square, not pointed as in hypertrophy; indeed, it retains the oval shape of the dullness of a normal heart. As dilatation occurs so frequently in emphysema of the lungs, the modification of the percussion sound must be remembered.

Auscultation.—The systolic sounds are short and sharp. They are high-pitched and resemble the diastolic. The latter may become enfeebled when the dilatation becomes excessive. The right and left first sounds may differ somewhat in intensity, and reduplication may occur. The sounds may be obscured by murmurs. The murmurs are due to previous valve disease or to incompetency on account of dilatation. The pulse is correspondingly small. In dilatation the alteration of the rhythm is extreme. There may be embryocardia, more frequently there is galloping rhythm of the heart. It must not be forgotten that as dilatation ensues, murmurs of various valve lesions may disappear, particularly the murmur of mitral stenosis. On the other hand, in the earlier stages particularly, murmurs develop on account of incompetence at the auriculoventricular orifices, in addition to the primary organic murmur. These murmurs may in turn disappear if the dilatation is controlled by careful treatment.

Adams-Stokes Syndrome.—A disorder characterized by extreme slowness of the pulse (heart-block) with syncopal and convulsive seizures as a result of actual ventricular stoppage. The syncope may occur in the course of partial heart-block (alteration of conductivity) or less frequently in complete heart-block: "Consequent upon influences which affect the pacemaker of the ventricle" (Lewis). The heart-block itself is the result of structural alterations in the bundle of His. The attack may vary from evanescent attacks of giddiness, and temporary unconsciousness, to actual convulsive seizures which may terminate in death, though by no means do all cases of heart-block have the seizures. The presumptive diagnosis of the condition depends upon polygraphic tracings for conformation.

Paroxysmal Tachycardia.—Alterations in the various functions of the heart have been discussed in the chapter dealing with pulse. One functional affection not yet discussed, presenting a definite clinical syndrome of paroxysmal attacks of tachycardia, is characterized by the sudden onset of rapid heart action associated with subjective sensations of oppression or tightness in the region of the heart, restlessness, weakness, and perhaps dyspnea. The pulse is rapid, often 200 per minute, and usually perfectly regular. The attack may last for several minutes to several days; they come on suddenly without cause and terminate as abruptly as they started.

The pathogenesis of the conditions is unknown. Lewis believes that premature auricular contractions, paroxysmal tachycardia, and auricular fibrillation are stages of the same process, and that the higher grades of a disorder are brought about, "either by an increase in the

exciting agent or by an increase in the irritability of the tissue responding to it." In the first two disorders the auricle beats in response to regular impulses generated in a single focus, in the latter to irregular impulses arising in various foci.

Diseases of the Coronary Arteries.—*Atheroma*, associated with the process in other vessels, or distinctly localized to the coronary arteries, affects these vessels. Its causal factors are those of endarteritis elsewhere. Its influence on the nutrition of the heart, either by sudden obstruction of the vessels by an embolus or by their gradual closure, with or without thrombus, is apparent.

Symptoms.—If an atheromatous coronary artery is suddenly obstructed by an embolus, death may be instantaneous. This is a common cause of sudden death.

If a thrombus forms, it is followed by anemic infarction, myocardial changes, and mural aneurism with the usual symptoms.

The onset of symptoms may be sudden, although the process (occlusion) has been in progress for an indefinite time. (1) Precordial oppression or dyspnea or angina pectoris may be the first indication. (2) Succeeding this, or immediately, dilatation of the heart, dyspnea, and venous stasis occur. The heart's action is persistently rapid and may be arrhythmical. (3) The presence of an aneurism may be made out. If there has not been previous valvulitis, no murmurs are heard until dilatation ensues. The patient may live three or four weeks, or as many months.

In a large group of cases occlusion is so gradual as to lead to myocardial changes only, with the attending symptoms of insufficiency.

Diagnosis.—Unfortunately, too often the diagnosis can only be provisional. Coronary artery disease may be suspected in sudden death if there has been a history of previous attacks of angina, if there is evidence of arterial disease elsewhere, and if dyspnea or anginoid symptoms preceded the fatal termination.

Thrombosis secondary to atheroma may be suspected if a patient, in whom there is no valvular disease, no pulmonary or renal disease, is seized with angina pectoris or dyspnea; providing tachycardia and arrhythmia follow, and in a short time cardiac dilatation, venous stasis, etc.

I have said elsewhere that a persistently rapid pulse uninfluenced by digitalis, indicates pericardial adhesion in the young; the same pulse uninfluenced by treatment points to coronary artery disease in the middle-aged and senile.

Angina Pectoris.—A disorder characterized by paroxysmal attacks of severe pain in the region of the heart, a sensation of pressure upon the chest, and fear of impending death. It is practically always associated with sclerosis of the first portion of the aorta and the coronary arteries and occurs usually in patients who have a generalized arteriosclerosis.

The nature of the condition is obscure. It has been attributed (1)

exhaustion of cardiac contractility; (2) to ischemia of the heart muscle from coronary stenosis or from spasm of the coronaries comparable to intermittent claudication; (3) to painful lesions of the first part of the aorta; (4) to neuritis of the cardiac nerves, or brachial plexus with the pain referred to the heart; (5) to a neurosis and (6) to acute dilatation of the left ventricle with secondary irritation of the sensory nerves of the heart.

Symptoms.—The attacks are usually induced by some extra work put upon the heart. It may be but slight. The pain is agonizing and is referred to the left shoulder and arm and neck. At times it is referred to the right side or to the abdomen. The paroxysm rarely lasts more than a minute. With it there is frequently hyperesthesia of the skin supplied by the second and third cervical segments, pallor and constriction of the peripheral arteries and profuse perspiration. The excruciating pain, the fear of death, and the feeling of constriction all cause the appearance of great mental anguish in the patient. True angina pectoris is usually a manifestation of serious myocardial disease, but may occur in other conditions, as aneurism of the aorta and disease of the aortic valves. The paroxysms may occur as follows:

(a) A person with myocardial degeneration may have one attack of angina terminating in death without premonitions of cardiac disease. They are the cases in which there is often found, associated with the myocarditis, extensive coronary artery disease, death being due to thrombosis.

(b) The paroxysms may continue for twenty-four to forty-eight hours before death closes the tragic scene. Arrhythmia frequently attends the breast pang. It may have preceded the angina for months or years. Dyspnea might also, although not necessarily, have been a forerunner, occurring either on exertion or at rest. It is not common to see the forms of dyspnea attended by shock and so often terminating fatally, associated. Nor do we usually see in these subjects of this rapidly terminating form of angina the cardiac asthma or dilatation, nor the nocturnal form of dyspnea, due probably to the same cause. We do see dyspnea from pulmonary congestion developing in the status. When the angina vera is replaced by angina sine dolore, we have asthmatic attacks in most of the cases. In rare instances the first paroxysm is followed by rapidly recurring attacks, increasing weakness of the heart, and death in cardiac asystole or the ingravescient asystole of Balfour. Houchard describes the condition as *l'état de mal angineux*.

(c) The paroxysms continue for several years. During the interval the patient will suffer, as in the preceding cases, from arrhythmia and the forms of dyspnea observed in that class. No other cardiac symptoms arise except in very rare instances. Pronounced dilatation may occur with the marked symptoms belonging thereto. On the other hand, it is usually the case that other symptoms or accidents of arteriosclerosis do not occur. Thus an anginal subject does not die of apoplexy; again, a prolonged intermission may occur in the attacks of pain, but

if arrhythmia has been present, it does not subside. The dyspnea, except on exertion (dyspnea of dilatation), may be in abeyance. Indeed, subsidence of the pain is usually due to moderate dilatation as indicated by the symptoms and physical signs.

(d) Occasionally we see angina sine dolore, at first described by Gairdner, the incident occurring in myocardial weakness with moderate dilatation.

Angina Pectoris Minor (Vasomotor Angina).—The so-called pseudo-angina occurs without organic lesions in neurotic, neurasthenic and hysterical individuals, usually women, and is considered to be of vasomotor origin. The attacks are frequently brought on by worry or injudicious eating; they are frequently nocturnal and precordial; the pain is not so severe as in true angina but more prolonged, and is improved by medication for the nervous system. Vasomotor phenomena (coldness, pallor, numbness, and weakness usually of the left arm, though at times of other portions of the body) are common. The condition is rarely fatal. A clinical similar *toxic angina* is sometimes seen following excesses in tea, coffee, and tobacco.

DISEASES OF THE ARTERIES

Arteriosclerosis (Arterio-capillary Fibrosis).—**Etiology.**—"Time, Tension and Toxins" (Osler). Arteriosclerosis occurs as the result of wear and tear of life and as the accompaniment of age. The time of its onset depends upon the quality of the arterial tissue which the individual inherited, and upon the amount of wear and tear. It may occur early in life, and entire families may show this tendency. Very frequently the sclerosis develops from exogenous and endogenous intoxication of the system, on account of which persistent spasm of the small vessels is set up; for blood of an impaired quality is passed with greater difficulty through the capillaries, as was taught by Bright. The blood tension is raised thereby. Alcohol, lead, and the poisons of gout and syphilis lead to this condition. The poisons of gout and the spirochetes of syphilis may set up directly an inflammation and degeneration of the arteries. In renal disease, arteriosclerosis is of common occurrence. The relation to the renal lesion differs. It may precede the kidney changes, or there may be a primary affection of the kidney.

Overfilling of the bloodvessels from excessive eating and drinking causes arteriosclerosis through constant overdistention of the vessels. In overwork of the vessels and excessive strain there is either heightened tension or increased peripheral resistance, the effect upon the bloodvessels being the same in either case. The result of the above causes alterations of the media followed by changes in the intima and adventitia, terminating in arteriosclerosis of the large arteries, or in arterio-capillary fibrosis of the smaller vessels. Involvement of the larger vessels

is usually nodular and patchy (atheroma); of the smaller vessels, diffuse, but usually associated with atheromatous areas in the larger vessels.

Symptoms.—The symptoms vary. They begin insidiously as a rule, and although arteriosclerosis may be present, the patient may die from other causes without even suffering from the effects of the disorder. The symptoms, when manifest, are referable to certain organs, according to the degree of sclerosis in them, and of these the cardiovascular symptoms naturally predominate. The early cardiac symptoms consist of dyspnea and palpitation upon exertion. Later, edema, cyanosis, and other signs of high-grade cardiac insufficiency appear. The left ventricle hypertrophies early in the course of sclerosis and presents the physical signs of the condition; a forcible impulse, extension of dulness downward to the left, a slightly muffled first sound at the apex, and a ringing accentuated aortic second sound. The first sound is frequently obscured by a murmur when there is much atheromatous roughing of the aorta. In the late stages of the condition dilatation of the heart takes place and with it, its train of symptoms and signs. The signs referable directly to the arteries consist of elevation of the blood-pressure (160 to 180 mm. Hg. or more), and palpable, frequently tortuous radial arteries. Symptoms, attributed to spasm of the various arteries or to consequent anemia of the part, consist of the pain of true angina pectoris, severe abdominal pain of short duration (abdominal angina), nocturnal muscle cramps, painful cramps of legs induced by walking and relieved by rest, and causing limping (intermittent claudication), and temporary transitory attacks of hemiplegia, monoplegia, or aphasia.

Cerebral symptoms, excluding those just mentioned, include irritability, loss of memory, headache, vertigo, insomnia, and tinnitus. Later symptoms referable to the cerebrum, include those of cerebral hemorrhage, thrombosis, and occlusion of the smaller vessels.

Renal symptoms of chronic interstitial nephritis are practically always present. Indeed, the sclerosis of the vessels, the hypertrophy of the heart, and the induration of the kidney are so closely related that in most cases the triad is aptly termed cardiovascular-renal disease.

Gastro-intestinal symptoms are those of "indigestion," although in many cases gastric ulcers are supposed to arise as the result of involvement of branches of the gastric arteries. Severe abdominal pain (abdominal angina), coming on several hours after eating, is attributed to angiospasm of sclerotic splanchnic vessels.

The *extremities* are usually but slightly involved. Pallor and coldness of the finger tips and toes, particularly if the patient is not using them, are frequent and early symptoms. Other signs are usually absent, although in a few cases gradual obliterations or thrombosis of the distal arteries causes gangrene, usually beginning in and involving one or both feet and gradually ascending. Erythromelalgia, Raynaud's disease, scleroderma, etc., are closely related in some way to arteriosclerosis, and may be an expression of the disease.

Diagnosis.—The diagnosis is suggested by the history and habits of the patient; confirmed by the findings of high blood-pressure, hardened peripheral arteries, hypertrophy of the heart, accentuation of the second aortic sound, and such changes in the urine as take place in chronic interstitial nephritis. The ophthalmoscope frequently shows changes in the retinal arteries before such changes are noted in the superficial vessels. Calcareous degeneration in the arteries of the extremities are beautifully shown by the x-ray. Arteriosclerosis in young people (under forty) is usually due to syphilis. It is usually first manifested as a syphilitic mesaortitis, because the first part of the aorta seems to be the site of predilection for the lodgement of the spirochetes. There is usually an associated involvement of the aortic valves, with resultant insufficiency, and a dilatation of the aorta. The process generally remains latent for years and may involve only the first part of the aorta. Sclerosis of the cerebral vessels, as a result of syphilis, causes the apoplexy that occurs in people under forty. Advanced arteriosclerosis is frequently associated with similar changes in the veins—phlebosclerosis.

Aneurism.—A true aneurism is formed by the distention of one or more of the arterial coats. It is usually saccular, but may be cylindrical or fusiform. The fusiform and saccular are the forms most commonly seen. A false aneurism follows wounds of the artery, causing a circumscribed hematoma. Dissecting aneurism arises from laceration of the internal coat of the artery. The blood dissects between the layers. Arteriovenous aneurism is seen when communication between an artery and a vein has been set up. If a sac intervenes, it is called a varicose aneurism. Sometimes communication is direct, the vein becoming dilated, tortuous, and pulsating. It is known as an aneurismal varix.

An aneurism may occur in the course of arterial sclerosis from diffuse distention of the coats. Its typical forms are seen in dilatation of the aorta, with one or more sacculated aneurisms on its surface.

Sacculated Aneurism.—This occurs from rupture of the media, independently of more extensive disease of the arteries, and in arteriosclerosis. The most common seat is the ascending portion of the aorta. It occurs early in the course of arteriosclerosis. Such form of aneurism is seen in the smaller vessels. Aneurisms also arise after the lodgement of an embolus permanently plugging the vessel, the proximal end of the vessel becoming dilated.

Aneurism of the Thoracic Aorta.—Syphilitic aortitis is the chief and important antecedent cause. Physical overwork, alcohol, gout, and the other causes of arteriosclerosis may also predispose to aneurism. It may be situated just beyond the aortic ring, at the junction of the ascending and transverse aorta, in the transverse, or at the beginning of the descending portion of the thoracic aorta. The larger aneurisms are at the two bends of the aorta.

SYMPTOMS.—The symptoms of aneurism are due to pressure and depend upon the position of the aneurism and the direction of its growth.

FIG. 186



Aneurism of ascending portion of arch of aorta. Tumor in first and second interspaces, extending into neck. Portion of sternum atrophied. (Original.)

Aneurisms, however, may exist without symptoms or appreciable physical signs. Even in a patient who has been under careful observation, sudden death may take place from rupture of a concealed aneurism, the presence of which had not been suspected during life. On the other hand, cases occur with characteristic pressure symptoms and with no physical signs.

Aneurisms of the ascending portion of the arch cause dislocation of the heart outward or toward the right pleural or forward. They

appear at the second or third right interspace, causing erosion of the ribs and sternum. The vena cava is compressed, causing enlargement of the veins of the head and arms; the subclavian vein may be compressed alone, causing enlargement and edema of the right arm. Localized edema may result, confined to the thorax. (See Edema.) If the aneurism is large, the inferior vena cava may be pressed upon, causing edema of the feet. The right laryngeal nerve may be involved, causing aphonia and dyspnea. Pain attends the aneurismal process.

Aneurism of the transverse portion of the aorta project below, forward, or backward. When forward, they produce tumors behind the manubrium, which from pressure cause destruction of the bone; if the aneurism projects backward, marked pressure symptoms are produced. When the trachea is pressed upon it causes dyspnea and cough which is paroxysmal. The esophagus may be pressed upon, causing dysphagia. The left recurrent laryngeal nerve may be pressed upon, causing paralysis of the corresponding vocal cord, with aphonia; or there may arise hoarseness or a peculiar monotonous quality and inability to reach a high note. Pressure on a bronchus may produce bronchorrhea and dilatation, which in turn may lead to localized abscess. The growth may extend upward, involving the coats of the innominate and carotid arteries on the right side, or carotid and subclavian on the left, markedly interfering with the pulse on both sides. Pressure on the sympathetic nerve is likely to take place in this situation, with contraction of one of the pupils, although at first it is sometimes dilated. The thoracic duct is sometimes compressed, leading to rapid wasting.

In the descending portion the pressure signs of aneurism are often not so marked. The vertebræ are likely to be pressed upon in this situation. The pain, therefore, is most intense. The esophagus and left bronchus are compressed. Dysphagia and bronchiectasis, the latter causing bronchorrhea with subsequent gangrene, are likely to occur. The cough and the fever in bronchorrhea, together with emaciation, simulate phthisis for which aneurism is often mistaken. In one of my cases, which had been treated for tuberculosis because of small hemorrhages, with the conditions above mentioned, death took place from rupture into the bronchus, causing sudden profuse hemorrhage. When the aneurism is adherent to the esophagus and slowly ulcerating into it, rupture may take place, followed by instantaneous death. The vertebræ may be eroded and symptoms of spinal compression arise.

SPECIAL SYMPTOMS.—Pain is an important and a constant symptom. It is sharp and lancinating, and may occur in paroxysms. It is more severe and constant when bone is eroded by pressure on the vertebræ, or on the ribs and sternum. The gnawing pain that attends ulceration of bone subsides after perforation has taken place. Anginal attack may attend the neuralgic pains just described. Pain sometimes follows the course of the nerves, extending down the arm or to the neck, or along the course of the intercostal nerves.

Cough.—The cough is peculiar. It is paroxysmal in many cases, and of a brazen, ringing character, indicating its laryngeal origin, due to pressure upon the recurrent laryngeal nerves. It is frequently paroxysmal when the pressure is directed upon the windpipe or bronchus. In the former instance the cough is dry, in the latter tracheal and bronchial. It is attended by a thin, watery expectoration which, if bronchiectasis with fermentation ensues, becomes thick and ropy. Dyspnea occurs more frequently in aneurism of the transverse portion, due (1) to pressure on the recurrent laryngeal nerves; (2) to compression of the trachea; (3) to compression of the left bronchus. Marked stridor

FIG. 187



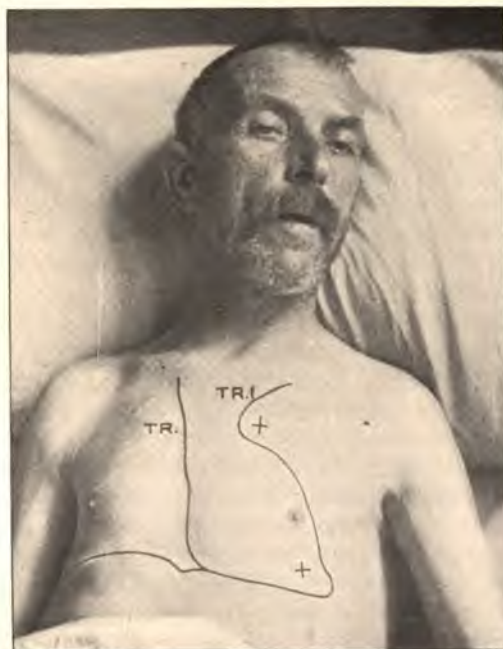
Aneurism of ascending and transverse portions of aorta projecting forward, destroying ribs and sternum. The skin ulcerated, and gradual external leakage took place. The bleeding continued in small amounts for a long time. (Original.)

attends the first form. When one of the recurrent laryngeal nerves, more particularly the left, is pressed upon, there is spasm or paralysis of the muscles of the vocal cord, causing hoarseness and loss of voice. Laryngoscopic examination should not be neglected, for paralysis of the abductor muscles without symptoms may be present.

Hemorrhage.—The hemorrhage may be gradual when there is slight leakage into the trachea at the point of compression. The amount of blood lost is small. It may take place externally (Fig. 187). Profuse hemorrhages, causing sudden death, occur from rupture into the trachea or bronchus, and from perforation into the lung. With regard to difficulty of deglutition, it may be said that the esophageal sound

visible tumor, pulsation and thrill may be felt in the suprasternal notch, if the head is bent forward, so that the tissues are relaxed, and the fingers pushed down toward the aorta. When the aneurism is filled or filling with clot, the tumor may be seen and felt, but no impulse will be transmitted to the hand, or thrill be felt by the fingers.

FIG. 188



Suspected aneurism. General endarteritis and valvulitis. TR, thrill and impulse +, murmur. Outline area of dulness; TR', in first interspace, thrill and murmur. (Original.)

Percussion.—Percussion furnishes the most reliable evidence of the presence of an aneurism in cases in which the tumor is not too deep-seated or small in size. The dulness may be relative only. The area of dulness is increased somewhere in the course of the aorta. It may be observed projecting outward at the right edge of the sternum when the ascending portion of the aorta is the seat of disease, or over the entire upper part of the sternum extending toward the left, when the transverse portion is diseased. It may be observed as an extension of cardiac dulness upward in the second and third interspaces. Sometimes dulness is detected in the scapular regions, particularly of the left side. The percussion tone is flat, and there is marked sense of resistance. Percussion must be employed with the patient in the upright and in the recumbent posture. During percussion of the

anterior chest, severe paroxysms of coughing, and the complaint on the part of the patient of severe pain over a localized area may be observed. The cough may also be induced by palpation of these areas.

The character of the note and the shape of the dulness must be noted at the end of full inspiration and of full expiration. Auscultatory percussion is of the utmost value. An aneurismal tumor may be present without thrill or murmur, but yields signs of dulness on percussion.

Auscultation.—Murmurs may not always be present. They depend upon the amount of coagulated blood in the sac. When present, the murmur is usually systolic in time, heard with maximum intensity, usually over the abnormal area of impulse or tumor, or over the increasing area of dulness. It is transmitted in the direction of the vessels, and may be heard louder in the vessels of the neck and along the course of the aorta. Often a double murmur is heard, the diastolic sound being frequently due to associated regurgitation at the aortic orifice. Sometimes the diastolic murmur alone may be heard. Increase in intensity or accentuation of the aortic second sound is pronounced. The sound is ringing in character, and is rarely absent in large murmurs.

THE PERIPHERAL VESSELS IN ANEURISM.—The pulse in the two radial arteries may show a marked difference both in volume and in time. The difference may indicate the position of the aneurism. If the pulse of the right radial is smaller than the left, the aneurism may be in or near the innominate artery; if the opposite, it is near or includes the orifice of the left subclavian. In the same way the difference in time may also aid in determining the location. Osler refers to obliteration of the pulse in the abdominal aorta and its branches. In one case he could not feel throbbing in the aorta and the femorals, although the circulation was unimpaired. The aneurism was in the descending portion of the aorta, and its pulsation was seen in the left scapular region. The sac was sufficiently large to act as a reservoir, which filled during the ventricular systole and emptied in a continuous instead of an intermittent stream, the effect of the ventricular systole being lost.

Tracheal Tugging.—Tracheal tugging may be obtained in one of two ways. By the old method the patient should be sitting or standing, while the observer sits or stands to one side, and faces him. With the hand farthest from the patient steadying the head, the observer gently but firmly grasps the surface of the cricoid cartilage, with the thumb and finger of the other hand, while the head is slightly thrown back. The head is then flexed, so that the neck is no longer stretched. The patient is then told to hold his breath completely, and any up-and-down movement of the trachea is immediately transmitted to the observer's fingers. One must not mistake the transmitted pulsation in the cervical vessels for such movement, and great care should be exercised to see that the breathing is entirely stopped.

In the other method the observer stands behind the patient, steadying the latter's head against his body, and the cricoid is firmly held between the tips of the first or middle fingers. The writer, after considerabl

experience, prefers this second method, on account of delicacy of touch, firmness of grasp, and comfort to the patient.

Similar to this, which is known as Oliver's sign, is Cardarelli's sign—lateral movement of the larynx. It differs from Oliver's only in the direction of the movement of the larynx, and is of similar diagnostic value. In some cases lateral or anteroposterior deviation of the trachea in the neck may be observed, and in other cases the upward and downward movement of the larynx and trachea during deglutition is much restricted. Hall has lately referred to a tracheal diastolic shock which he regards as important in the diagnosis of aneurism.

DIAGNOSIS.—The special points of diagnosis are: the etiological factors; the antecedent pathological conditions, as arterial sclerosis; the occurrence of pain and pressure symptoms; and the physical signs. These have been sufficiently dwelt upon, and it is not necessary to again consider them. It must not be forgotten that aneurism may be present without diagnostic physical signs, and, on the other hand, the pressure symptoms may also be in abeyance. If one of the two is present in a male subject past forty, with a previous history of syphilis, gout, alcoholism, or muscular strain, the probability is that an aneurism exists.

Cases are often seen that present physical signs of aneurism which are due to valvulitis and occasionally to pericarditis.

The pressure symptoms always point to some form of intrathoracic disease as the cause of the group of symptoms. Thus in cancerous disease of the lymphatic glands, or other tumors within the mediastinum, pressure symptoms exactly simulating aneurism may be present and also the physical signs of a tumor. In tumor, however, there is usually absence of certain etiological factors of aneurism, such as strain, syphilis, etc., and the tumor, if primary, is very common in young subjects. The tumor rarely projects externally, and still more rarely pulsates. If pulsation is present, it is not of the expansile character seen in aneurism, nor is there as decided a systolic shock when the ear is held against the chest. By the same method we observe the shock of the heart-sounds, which are notably lessened or absent in tumors from causes other than aneurism. In deep-seated tumors with pressure symptoms the condition of the arteries, apart from aneurism, is of diagnostic importance. Accentuation of the aortic second sound, with hypertrophy of the heart, points to aneurism. The presence of tracheal tugging is also a valuable diagnostic point in its favor. In tumor, and especially in cancer, there are emaciation and the development of a cachexia, which, as is well known, is most pronounced in cancer of the esophagus. Most intrathoracic tumors are fatal within a year of the onset of symptoms, while aneurism may last a long time. Cancer of the esophagus, from its frequent point of election near the left bronchus, often simulates the pressure symptoms of aneurism.

Aneurism must be distinguished from the pulsation of the aorta

CHAPTER XXXIX

DISEASES OF THE MOUTH, THE TONGUE, THE PHARYNX, AND ESOPHAGUS

THE MOUTH

THE predisposing causes of disease of the mouth are infancy, dentition, decayed teeth, poor nutrition, unsanitary conditions, wasting diseases, and trauma. The exciting causes are microorganisms. Chronic suppurative processes of the mouth, as pyorrhea alveolaris, gingivitis, carious teeth, etc., are frequently the cause not only of poor health and anemia from absorption of toxins, but may result in specific disease, as acute gastritis or enteritis, from the constant swallowing of the pus.

Stomatitis.—Catarrhal Stomatitis.—This develops most frequently in children, during dentition or from gastro-intestinal disturbance.

Symptoms.—The mucous membrane of the mouth is swollen, red, and hard. The inflammation may spread to the gums, causing tenderness; to the tongue, which becomes red, swollen, and coated; or to the salivary glands, causing salivation.

Aphthous Stomatitis.—This presents similar symptoms plus the appearance of small yellowish-white spots dotted over the mucous membrane of the mouth. These little vesicles break down into shallow, sensitive, superficial, grayish ulcers, with raised red margins. There is associated fever, unwillingness of the child to take food, and a fetid breath.

In foot-and-mouth disease, aphthous ulceration is seen, but the local and general symptoms are decidedly more pronounced than in the simple form.

Ulcerative Stomatitis.—This occurs in ill-nourished subjects, and is often intercurrent with wasting disease. It may be seen in epidemic forms in camps and penal institutions. The inflammation is chiefly confined to the gums, which are swollen, red, and covered with yellowish, linear ulcers. The ulcers are usually at the gingival border. The flow of saliva is much increased, and it is acid in reaction. The fetor of the mouth is great. The submaxillary glands are enlarged.

Parasitic Stomatitis.—Thrush.—An inflammation characterized by the production of raised white patches upon the mucous membranes, looking like small curds of milk. The patches vary in size, and are discrete at first, later they coalesce. Portions of the patches can be readily removed, and if there is a question as to diagnosis, the causative organisms, the *Odium albicans*, can be readily found by microscopic examination.

Gangrenous Stomatitis.—Noma.—This is an inflammatory process characterized by a rapidly spreading gangrene of the gums, alveolar processes, and cheeks. It occurs usually secondary to the acute diseases of childhood, particularly measles and scarlet fever. A small indurated dark red spot first appears, which rapidly increases in size and soon ulcerates. Rapid necrosis of the surrounding tissue takes place, so that the cheek may be perforated and the jaw involved. The characteristic odor of gangrene attends the process. Prostration, fever, and other constitutional symptoms are marked.

Mercurial Stomatitis or Ptyalism.—This is an early manifestation of mercurial poisoning. It particularly affects the gums and the salivary glands. The gums are swollen, red, sore, and bleed on the slightest touch. The teeth become loosened; the breath is fetid and offensive; the tongue is swollen and coated, and there is profuse salivation. The earliest symptoms, a metallic taste in the mouth and pain on mastication, are warning signals of poisoning by too large doses of mercury or indicate undue susceptibility to the drug.

Other Varieties of Stomatitis.—Leprosy, glanders, and actinomycosis are associated with stomatitis, and are recognized by the presence of the characteristic organisms and the associated symptoms of the disease elsewhere.

Ulcers of the Mouth. *Solitary ulcers* are seen in herpes; in *stomatitis materna* affecting the lips or cheeks of nursing women; in *syphilis* (mucous patches) and in a few other conditions in which the mouth involvement is of minor importance.

THE TONGUE

Acute Glossitis. This results most frequently from trauma, burns, insect bites, or from self-biting during a epileptic convulsion. It is usually unilateral (hemiglossitis). The tongue is swollen, tender, heavy, and coated. Salivation, inability to speak, dysphagia, and occasionally dyspnea are direct results of the inflammatory process. The condition terminates in suppuration in about one-third of the cases. When this occurs, constitutional symptoms are pronounced.

Chronic Glossitis.—A chronic superficial inflammation characterized by pain, reddening, and fissuring of the tongue is occasionally seen.

Leukoplakia.—This is a chronic disease of the mucous membrane of the tongue, characterized by smooth pearly patches of thickened epidermis, appearing on the sides of the organ.

Eczema of the Tongue.—This is a condition characterized by desquamation of the superficial epithelium of the tongue in rounded patches, the periphery spreading while the central portions heal. When extensive the tongue may be covered by these patches, resembling a geographical map (geographical tongue).

Syphilitic Lesions of the Tongue.—These may be found in any of the three stages of the disease. The tongue is one of the most frequen

sites of a chancre in extragenital syphilis. Mucus patches are found on the tongue as well as the cheek or lips in the secondary stage. The tertiary lesions are those of gummata.

Tuberculosis.—Tuberculosis of the tongue is usually manifested by submucous tuberculomata which eventually ulcerate.

Atrophy.—Atrophy of the tongue is a rare condition. Hemiatrophy occurs as a result of disease of the nerves or centres supplying the tongue.

Hypertrophy.—Hypertrophy of the tongue, non-inflammatory, or macroglossia is generally congenital, and is seen in idiots, cretins or in myxedema.

Angina Ludovici.—This is a streptococcic cellulitis of the tissues under the tongue and forming the floor of the mouth. It is characterized by sudden onset, wooden and rapidly spreading induration of the sublingual tissue, which may extend from the ramus of the jaws to the cheeks and eyes; reddening, edema, and tenseness of the skin under the jaw; and pronounced constitutional symptoms.

THE PHARYNX AND TONSILS

The pharynx and tonsils are lined with mucous membrane which is subject to the same diseases, especially inflammation, which cause the same general symptoms as affect mucous membranes in general. The large muscles of the pharynx are subject to affections which belong to muscular tissue generally, hence *rheumatism* or *paralysis* in particular may occur. The tonsils from their anatomical position are subject to mild trauma and to the constant action of septic material, while their cryptic and irregular anatomical arrangement affords splendid lodging-places for the implantation and growth of bacteria. Pathogenic bacteria frequently invade the tonsil, which acts as a portal of entry for toxins of microorganisms in such diseases as acute articular rheumatism, chorea, acute endocarditis, acute nephritis, glandular tuberculosis, and diphtheria. Chronic tonsillar infections may cause, as a result either of repeated entry of specific organism of a mild type, or as a result of elaboration and absorption of toxins, such conditions as are found in the chronic arthritides. The symptoms of pharyngeal and tonsillar disease are the result of their anatomical relations. *Difficult and painful deglutition* is the most characteristic local symptom. *Dyspnea* occasionally is present if there is sufficient change to cause obstruction to the entrance of air into the lungs. *Cough* also may be an occasional subjective symptom of pharyngeal diseases. The general symptoms are those of the causative disease or pathological process.

Acute Follicular Tonsillitis.—For convenience the catarrhal, erythematous and herpetic forms of tonsillitis are grouped under this one head. The common exciting cause of acute tonsillitis is a streptococcic infection of the tonsil. Predisposing factors are previous attacks,

exposure to cold, fatigue, inhalation of noxious emanations, and season (Spring). At times the condition appears to be mildly contagious, and numerous epidemics of tonsillitis have been reported, often following the drinking of infected milk.

Symptoms.—The onset is sudden, associated with chills or chilly sensations, a rapid rise in pulse-rate and temperature (103° to 105° F.), backache, malaise, and prostration. There is also difficulty in deglutition, hoarseness of the voice, fetor of the breath, and "sore throat." Usually both tonsils are enlarged and dotted with small bluish-white spots, which tend to coalesce. The tongue is heavily coated and the cervical glands are enlarged. There is a leukocytosis. The urine is loaded with urates and shows a trace of albumin; rarely an acute nephritis may develop, with pronounced albuminuria and casts. The acute symptoms subside in twenty-four to forty-eight hours, and the fever departs in four or five days as a rule.

Diagnosis.—Acute follicular tonsillitis may simulate diphtheria if a distinct pseudomembrane is formed on the tonsil, but this membrane is localized while the diphtheritic membrane spreads to the pillars or may be seen on the uvula; furthermore, it can be readily picked off while a diphtheritic membrane is adherent and removal of a portion of it causes bleeding and erosion of the mucous membrane. In doubtful cases a culture should always be made.

Suppurative Tonsillitis.—**Quinsy.**—This is most common in adolescence, and results from the same causes as the follicular form, but ends in abscess formation.

Symptoms.—The constitutional disturbance is great, and there is high fever, rapid rise of the pulse, and great prostration, associated at times with slight delirium. The soreness of the throat soon becomes intense, and in twenty-four hours, deglutition becomes impossible. At the end of forty-eight hours the patient is barely able to open the mouth; salivation is pronounced; and the voice is nasal or suppressed. If the mouth can be opened sufficiently, one or both tonsils are found to be enlarged, hard and tense, and often extending out to the midline; the peritonsillar tissue is red and edematous; and the cervical glands are swollen. In three to six days suppuration ensues and the tissue above and in front rather than the gland itself will be found to fluctuate. Unless there is operative intervention the abscess will rupture spontaneously into the mouth, affording instant relief. Rarely rupture into the pharynx with the entrance of pus into the larynx may cause death from suffocation, or the abscess may open into the carotid artery.

Diagnosis.—The characteristic pain and difficulty in swallowing should distinguish the condition from tetanus.

Hypertrophied Tonsils and Adenoid Vegetations of the Nasopharynx

—These two conditions, due to hypertrophy of the lymphoid tissue of the throat, are usually associated, occurring to a greater or degree in practically all children between the ages of four and four, and may result from chronic inflammation of the nasopharynx, ↓

the acute exanthemata, as a result of general lymphatic enlargement (lymphatism) or as an apparently inexplicable condition.

Symptoms.—Mouth-breathing is the cardinal symptom. In the severer cases there is some discharge from the nose and the hearing is frequently impaired. The child is restless at night and it snores and breathes irregularly; enuresis is a frequent associate symptom. Attacks of indigestion are common. Headache, listlessness and indisposition for mental exertion are marked. The patients are backward in their studies and unable to concentrate upon any subject (aprosixia); choreiform spasm of the face often occurs in connection with the condition. The voice is thick and muffled and cough is often present. The expression of the face is characteristically dull and stupid. The mouth is open in breathing, the lips are thickened and dry, and the nostrils flattened laterally. There is general lack of physical development. The chest presents certain deformities of which the so-called "chicken-breast" is most frequent, although the chest may be rounded and barrel-shaped as a result of emphysema, or there may be a "funnel-breast."

Diagnosis.—The diagnosis is based upon the characteristic mouth-breathing and facies of the patient. The enlarged faucial tonsils are seen, while in small children the small irregular adenoid bodies in the nasopharynx are easily palpated.

Acute Pharyngitis.—Acute pharyngitis or *sore throat* follows cold or exposure. The acute inflammation may be associated with rheumatism or gout. The local symptoms are dysphagia, dryness of the throat, constant desire to hawk and cough, hoarseness, and stiffness of the neck. The general symptoms are not marked. The attack is ushered in by chilliness and slight fever. On examination the mucous membrane is congested, dry, glistening, swollen, and covered in spots with sticky secretions.

Phlegmonous Inflammation of the Pharynx.—This is a rare occurrence. There is severe dyspnea, extreme dysphagia, and pronounced

FIG. 189



Appearance in adenoid disease.
(Dawson-Williams)

constitutional reaction. The inflammation invades the larynx and trachea and surrounding structures. Death occurs in a short time from exhaustion.

Chronic Pharyngitis.—The condition follows acute attacks and is a frequent accompaniment of nasal catarrh. It is common in smokers, alcoholic subjects, and those accustomed to loud speaking, as clergymen and auctioneers. It frequently attends indigestion. Examination shows a hyperemia of the mucous membrane which may be studded with millet-seed projections (enlarged follicles), or is dry and glistening.

Retropharyngeal Abscess.—Abscess in the connective tissue of the posterior pharyngeal walls occurs most frequently in healthy infants. More rarely it occurs as a sequel of the infectious fevers or results from caries of the cervical vertebræ. The symptoms are high fever, alteration in the voice, dysphagia, impeded breathing, stiffness of the neck and enlarged glands. A projection into the pharynx can be seen or distinctly felt. In the latter case fluctuation may be demonstrated.

THE SALIVARY GLANDS

Ptyalism.—(See page 656.)

Xerostomia.—A rare condition, characterized by the diminution or cessation of salivary secretion is occasionally found, usually, in neurotic women. The mucous membrane of the mouth and tongue is red, cracked, dry and glistening. Deglutition, mastication, and speech are interfered with.

Specific Parotitis.—(See Mumps, page 469.)

Symptomatic Parotitis.—This inflammation of the parotid gland arises in the course of acute infectious diseases, particularly typhoid fever, or may be the result of injury, operation, or disease of the abdomen or the pelvis, especially of the genito-urinary organs. The symptoms are acute swelling, redness, and heat over the gland, associated with painful and difficult deglutition. Suppuration ensues in a certain proportion of cases.

Chronic Parotitis.—The condition usually succeeds chronic inflammation of the throat. Both parotid glands and the submaxillary glands as well are usually enlarged.

Mikulicz's Disease.—The condition is characterized by chronic, painless inflammatory enlargement of the lacrymal and salivary glands.

Salivary Calculi.—These stones are usually found in the rarely in the gland. The characteristic symptom is intermission in the floor of the mouth. A small sound introduced into W duct, the usual site, will verify the diagnosis if the hard, smooth of the stone is felt.

Tumors of the Salivary Glands.—Gaseous tumors of the gland and Steno's duct are due to the entrance of air into

They may attain the size of a walnut. The condition usually occurs in glass-blowers and players of wind instruments.

Mixed Tumors.—These in three-fourths of the cases affect the parotid. They are slow-growing, movable, smooth tumors, which often reach huge proportions.

THE ESOPHAGUS

Acute Esophagitis.—Acute inflammation of the esophagus occurs (1) as a result of mechanical irritation by foreign bodies or very hot liquids or of chemical irritation by strong corrosives; (2) secondarily from extension of catarrhal processes of the pharynx and stomach or as a result of cancer of the esophagus; (3) occasionally in the course of diphtheria, typhoid fever, pneumonia, measles, and pyemia.

Symptoms.—The main symptom is pain, which is substernal, continuously dull and aching until swallowing is attempted, when it becomes intense. There is often tenderness along the course of the tube with extreme thirst and regurgitation of frothy, glairy, often bloody mucus. The *diagnosis* is usually based on the history and the occurrence of severe dysphagia. The use of sounds is contra-indicated. When the lesion is due to a foreign body, there is spasm of the gullet with regurgitation of food. The swallowing of corrosives causes easily recognized eschars of the mouth and is often marked by profound prostration. It is likely to lead to stricture as a result of cicatricial contraction.

Chronic Esophagitis.—Pain and difficulty in swallowing solids and the expectoration of large amounts of viscid mucus are the characteristic indications of a chronic inflammation of the esophagus.

Ulcers of the Esophagus.—Ulcers of the esophagus may be the result of acute inflammations, cancer, syphilis, or typhoid fever. *Decubital*, *pressure*, and *peptic ulcers* near the cardia are occasionally found. The symptoms are localized pain and pain on swallowing, associated at times with vomiting of blood. The diagnosis can be verified by the use of the esophagoscope.

Stricture of the Esophagus.—Stricture is due to contraction of a healed ulcer, congenital stenosis, external pressure, and cancer of the wall. The symptoms are progressive difficulty in swallowing, regurgitation of alkaline masticated food, and very gradual loss of weight and strength. The diagnosis is assured and the stricture definitely localized if the stomach-tube or an esophageal bougie, a long piece of flexible whalebone with an acorn-shaped head, can be passed only a certain distance.

Cancer of the Esophagus.—The growth usually involves the lower third of the gullet. It results in stenosis, with dilatation and hypertrophy of the tube above the narrowing.

Symptoms.—Progressive dysphagia often becoming extreme, rapid emaciation, slight fever, enlargement of the cervical glands, cough,

and the regurgitation of food at once if the cancer is in the upper portion of the esophagus; in fifteen to twenty minutes, if it is in the lower portion. Perforation may occur into the air passages, resulting in immediate cough and dyspnea with the later development of the symptoms of pulmonary gangrene, abscess or aspiration pneumonia; or into the aorta, mediastinum, or pericardium.

Diagnosis.—Rapid and progressive loss of weight with increasing dysphagia in a person over fifty is most suggestive of cancer of the esophagus. If bleeding occurs after the passage of a sound or if fragments of cancer tissue are brought up by the stomach-tube the tentative diagnosis is confirmed. Esophagoscopy examination yields a positive diagnosis. A benign stenosis is differentiated from a malignant stenosis by the history, if there is a stricture, or by the physical findings, if there is stenosis from pressure outside the tube.

Dilatation and Diverticula of the Esophagus.—Symmetrical dilatation of the esophagus occurs above a stricture of the tube. Idiopathic dilatation is a rare condition. The chief symptom is the habitual regurgitation of alkaline undigested food. A sensation of distention along the course of the esophagus, with heat and burning, may at times be noticed by the patient.

Asymmetrical dilatations or diverticula of the esophagus results either from frequent pressure within the tube by large, poorly masticated boli of food or from the traction of inflammatory adhesions from bronchial lymph glands upon the external anterior surface of the gullet. Traction diverticula are small, symptomless as a rule, and may be situated anywhere along the course of the tube. Pressure diverticula are usually situated high up in the posterior wall at the junction of the pharynx and esophagus. They may attain a size so enormous that a palpable tumor can be felt in the neck. As a result of the saccular form of the diverticula, portions of food are retained for a time sufficient to become decomposed. This causes a foul breath, and at times results in ulceration of the tube. The characteristic complaint is regurgitation of food during the act of eating. The regurgitated food may contain offensive remnants of the food taken at a previous meal which had remained in the pouch. Bougies and sounds, unless they are bent, will readily pass small sacculations. Large sacculations, particularly if filled, usually catch the sound and prevent its further progress. The x-ray, after a bismuth meal, will usually diagnosticate the condition.

Rupture of the Esophagus.—This occurs in rare instances as a result of disease of the walls or from violent vomiting. It causes extreme pain, hemorrhage from the mouth or bowel, shock and death.

Neuroses of the Esophagus.—These occur most frequently in hysterical hypochondriasis, and neurotic conditions or in the course of hydrophobia, epilepsy, or chorea.

Sensory Neuroses.—These are manifested most frequently by a sensation of a lump in the esophagus (oesophagus).

Motor Neuroses.—These are characterized by spasm of the esophagus (*esophagismus*). The symptoms are sudden inability to swallow solid foods, persisting for a variable time and regurgitation of food during the attack. Emaciation and the other grave effects of organic disease are absent.

The diagnosis is readily confirmed by the passage of a bougie which may meet an apparent obstruction, but gradually slips through it in a minute or two.

Paralysis.—This results from diphtheria or from central disease, especially bulbar palsy. Difficulty of deglutition is the main symptom.

CHAPTER XL

DISEASES OF THE PERITONEUM AND RETROPERITONEAL GLANDS, THE STOMACH, AND INTESTINES

DISEASES OF THE PERITONEUM

Ascites.—An accumulation of fluid in the peritoneal cavity may be due to local or general causes.

The local causes are chronic peritonitis, portal obstruction, particularly from cirrhosis of the liver, and abdominal tumors, notably solid ovarian tumors.

The general causes are those that give rise to dropsy. It is well to remember that ascites may occur in heart disease without the preliminary production of edema of the lower extremities.

Inspection.—If the effusion is large the skin is tense; lineæ albicantes are present; there is pouting of the umbilicus, dilatation of the superficial veins and fulness of the dependent parts. If the effusion is very great the abdomen is rounded and barrel-shaped.

Palpation.—Fluctuation is present, demonstrated by placing one hand on the flanks and lightly tapping the opposite side. The vibration of the abdominal wall is avoided by having an assistant place his hand vertically in the midline. The position of solid organs is determined by *dipping down* gently with the tips of the fingers over the surface of the organ sought for.

Percussion.—Dulness is present over the fluid which gravitates to the most dependent parts, and hence changes in the location of the dulness occur as the position of the patient is changed.

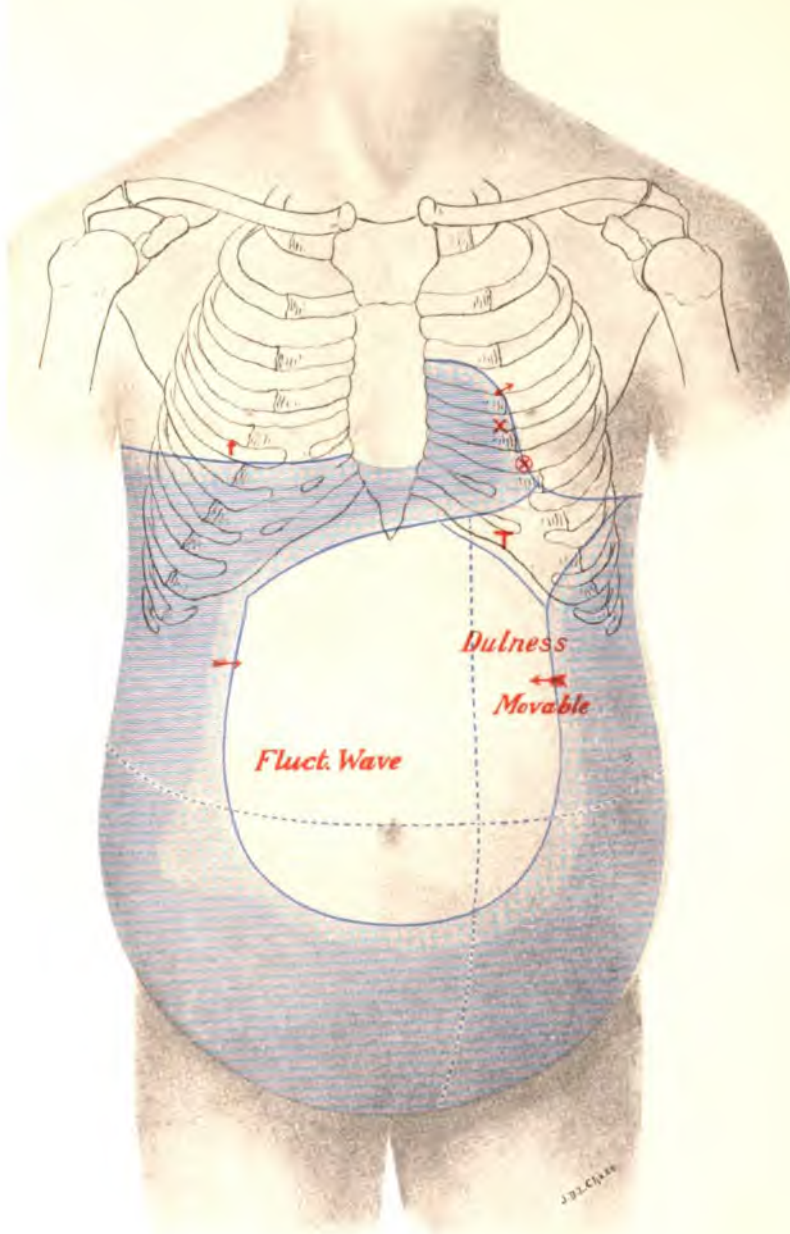
Aspiration.—The character of the fluid is ascertained by aspiration, a most important procedure in arriving at a diagnosis of the cause of the ascites. (See Chapter XXIX, page 445.)

Differential Diagnosis.—Ascites must be distinguished from cysts of the ovary and pancreas, and a hydatid cyst. Pregnancy, distended bladder or large lipomata are not likely to be confused with ascites with its movable dulness.

Peritonitis.—Inflammation of the peritoneum may be acute or chronic, general or localized, primary or secondary.

Acute General Peritonitis.—This condition usually occurs secondarily to inflammation of one of the peritoneum-covered viscera, as a result of rupture of an organ into the peritoneal cavity, *e. g.*, perforating gastric ulcer, or after operation. It occasionally occurs as a terminal infection

PLATE XXIII



Ascites.

Blue shading shows level of dulness in recumbent posture.
Dotted lines indicate change of level of fluid in other postures.

kidney disease, gout, or arteriosclerosis. So-called idiopathic may arise in the course of an attack of rheumatism or after

FIG. 190



Bimanual palpation to determine the presence of fluctuation.

exposure to cold. Whatever the cause of peritonitis it is always the result of the entrance of pyogenic bacteria into the peritoneal cavity, most frequently the *B. coli communis* or some strain of streptococcus.

FIG. 191



Ascites. Upper limits of dulness indicated by the dotted line. Umbilicus prominent. (Original.)

SYMPTOMS.—The onset is usually rapid, associated with chills and rigor. Pain is an early symptom localized at first over the site of the

of great interest
subject to
or speaking
prostration and
general type. I
or 100° F. The
of shock after pe
of poor volume
wiry characteris
philes except in th
resistance. Vomiti
and the extension
expulsive and con
stained and watery
disease have a fecal
replaced by a simple
stant for as long as tw
constipated as a result
Frequent and painful
although retention may

Physical Signs.—The
assumes the dorsal decub
relieve the tension of the
shallow and hurried. The
the features pinched. The
wasted and signs of collapse
distended.

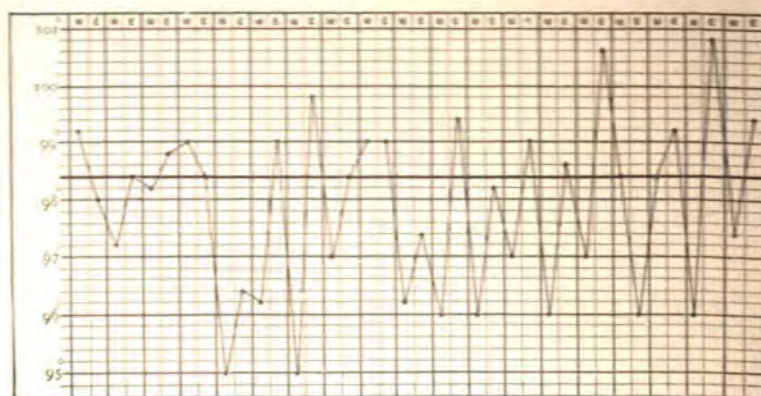
Inspection shows an ex
tended-like rigidity. Percus
anterior abdominal wall. In
cases some distension. No
one from distension decreases, and
change of normal percussion
shows the abdomen.

Palpation.—The chief difficulty
in examining the pelvis with
the hand is the rigidity of the
abdominal wall. It may be possible to
feel the uterus, but often by the
vaginal route. Peritonitis
may be such that he does not
and a leukocytosis are of
acute hemorrhage
and a surgical intervention for dis

the pleura and pericardium, multiple progressive serositis or Concato's disease, or in association with arteriosclerosis and granular kidney. The chief diagnostic feature of the condition is a persistent and severe ascites.

Tuberculosis of the Peritoneum.—Tuberculosis of the peritoneum may be acute or chronic, usually the latter form. It may develop during the course of acute miliary tuberculosis, and give no characteristic symptoms of peritonitis, the miliary tubercles being recognized only at autopsy. It may occur secondarily to tuberculosis of the lung or intestine, and more rarely secondary to tuberculosis of the Fallopian tubes, prostate, seminal vesicles, or lymph glands. It may run a very acute course, simulating typhoid fever, or may be protracted and only moderately severe.

FIG. 192



Tuberculous peritonitis. Subnormal temperature. (Original.)

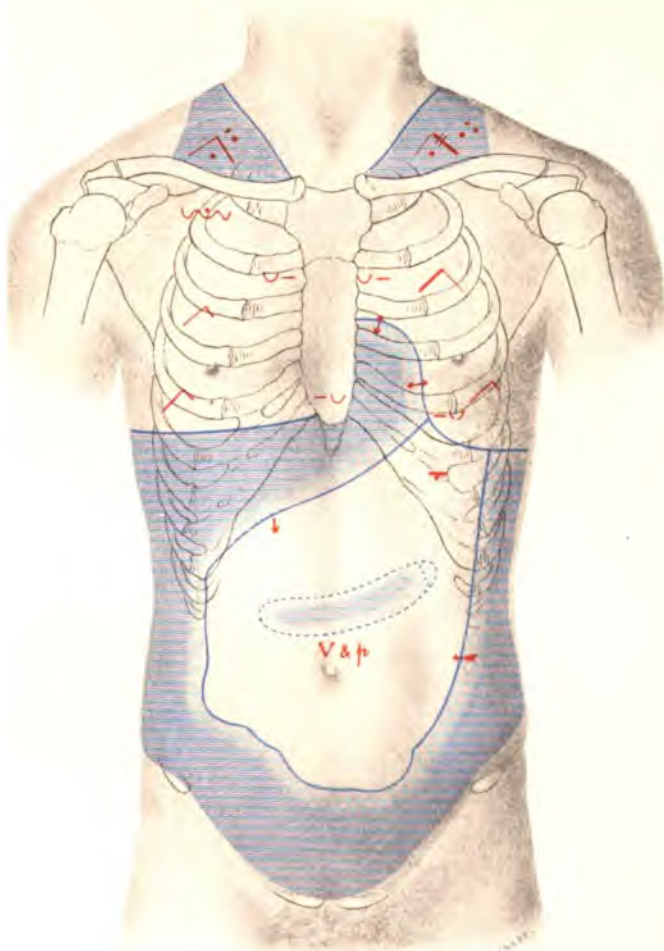
The constitutional symptoms are those of tuberculosis elsewhere, although usually more severe. There is an irregular temperature curve which may be continuous or remitting. Emaciation is usually rapid, and severe sweating is a characteristic of the disease. The bowels are constipated and there is frequent vomiting. The local physical signs may be divided into four classes as they exhibit various differences.

1. **Enlargement of the Abdomen with Effusion.**—Here the signs are those of moderate ascites. Adhesions are usually few in this form.

2. **Enlargement with Tumors.**—The tumor or masses are due to adherent coils of intestines, the rolled-up omentum or enlarged mesenteric glands. They are usually always found in the upper abdomen, are movable, and vary with the position of the patient. They are doughy in feeling, and percussion over them usually gives a muffled resonance.

3. **Enlargement with Tumor and Fluid.**—Here the signs are those of the fluid and the tumors, which are usually not as well-defined as in group two.

PLATE XXIV



Tuberculosis of the Peritoneum.

sinual exudate (not freely movable); omental tumor. Consolidation at apices.

4. **Obliterative.**—Early in this form there may be some enlargement, but the universal adhesions soon retract the abdominal wall, which has a peculiar induration and is usually somewhat tender. Percussion gives a modified tympany.

Diagnosis.—Tuberculosis of the peritoneum may simulate typhoid fever if the temperature is continuous. They may often only be differentiated by a blood culture or the serum reaction.

The tuberculous masses may be mistaken for cancer in old people. If the fluid is sacculated it may be confused with ovarian or other intra-abdominal cysts. The ascitic form of peritoneal tuberculosis may be confused for a time with cirrhosis of the liver. Tuberculosis of the peritoneum, if sudden in onset, may be only differentiated from acute peritonitis by the absence of any history of a cause for the condition. It may resemble acute appendicitis in the beginning of the

FIG. 193



Tuberculous peritonitis. Note the emaciation, the length of the arm, the enlargement of the wrist, the size of the hand, and the tapering fingers.

attack. In both cases the blood-count will aid in the differentiation. The diagnosis can be materially aided by noting the occurrence of irregular temperature and by the demonstration of tuberculosis of other organs. In some cases the diagnosis can be made by performing paracentesis and then studying the character of the fluid withdrawn. At other times resource must be had to opening the abdomen.

Tumors of the Peritoneum.—These may be malignant or non-malignant. The non-malignant tumors may be solid (fibroma, myoma, or lipoma) or cystic (mesenteric, hydatid, omental, or dermoid). They present no characteristic symptoms or signs. The malignant tumors may be rarely primary, as *endothelioma*, or much more frequently, secondary to cancer elsewhere. *Cancer* occurs late in life. Pain is a frequent occurrence. Ascites without evident cause in an old person, and a localized tumor without fever, are strongly suggestive of carcinoma of the peritoneum. The ascitic fluid is usually blood-stained. *Retro-*

a large number of patients (75 per cent.), while a hyperacidity often occurs in the early stages. Examination of the fasting morning stomach contents has frequently demonstrated tubercle bacilli swallowed during the night and which had not previously been found in the sputum.

Anemia or Chlorosis.—Gastric analysis in these conditions usually show a subacidity or an actual achylia with increased gastric motility.

Diseases of the Heart.—Chronic digestive symptoms the result of venous congestion of the stomach often call attention to unsuspected cardiac disease.

Diseases of the Kidney.—Vomiting is often an early symptom of chronic nephritis and uremia. It may occur before other subjective or objective symptoms of the condition. A movable kidney causes symptoms from pressure on the pylorus and reflexly from its influence on the nervous system.

Diseases of the Liver.—The liver is in such close relationship to the stomach that disturbance of its function usually causes a disturbance in the stomach with appropriate symptoms. The same generalization applies to the gall-bladder.

Diseases of Metabolism.—*Gout.*—It is well to remember that gastric disturbances are more likely to occur in gouty individuals than in normal ones. Sensory stomach symptoms associated with hyperacidity are found in this condition.

Diabetes.—Gastric symptoms in this condition are the exception rather than the rule, though occasionally a patient will consult a physician for symptoms such as excessive thirst, anorexia, or constipation, without knowledge of the underlying cause. A hypoacidity is usually present.

Diseases of the Intestines.—As with the liver, the close relationship of the intestines to the stomach frequently makes the diagnosis of the primary disease extremely difficult.

Diseases of the Pancreas.—In acute hemorrhagic pancreatitis the location and duration of the pain may be such as to suggest a perforating peptic ulcer.

Diseases of the Nervous System.—Locomotor ataxia may have as a first symptom gastric crises. A mild gastralgia and indigestion may also mark the onset of the condition. Vomiting may be the initial symptom of cerebral tumor.

Menstruation.—Secretory and motor changes are usually present during the menses. In pregnancy an anacidity is often found lasting for several weeks before and after parturition. At the climacteric various functional disturbances arise.

General Examination.—It has thus been shown that the stomach may be the primary cause of a secondary disease; or it may itself be secondarily involved with resulting organic or functional changes; or finally reflex symptoms, due to its close relation to the sympathetic system, may arise, as in a cerebellar tumor. Therefore, subjective symptoms of disease of the stomach should be carefully analyzed.

Thorough physical and laboratory examinations should be made before coming to any conclusions as to the primary pathological change causing gastric symptoms.

The analysis of the gastric contents is of primary and particular importance, but only slightly less important is the complete blood count, the urine analysis, examination of the feces, and in some cases the x-ray examination.

Organic Diseases of the Stomach.—Acute Catarrhal Gastritis.—The simple variety of gastritis varies in degree from a slight attack of nausea and vomiting to a severe form associated with a pronounced constitutional reaction. The onset is sudden, following indiscretions in diet. Anorexia is present, associated with nausea and vomiting. There is a sense of fulness and often colicky pain in the epigastrium. The vomiting of the offending food may relieve the condition or it may continue with the vomiting later of bile, mucus, and gastric secretion. Usually a few hours after the onset diarrhea occurs. Examination of the stomach will show some epigastric tenderness. The analysis of the vomitus shows a diminution or absence of hydrochloric acid and the presence of organic acids.

Diagnosis.—If adequate cause for the symptoms cannot be discovered, then the patient should be examined most thoroughly. The acute exanthemata frequently commence with symptoms of a gastritis; therefore until the time of pathognomonic symptoms has passed, the patient, children especially, should be carefully observed if the gastritis persists. The same can be said of diphtheria, dysentery, pyemia, meningitis, and pneumonia. A histological study of the stomach of patients dying as a result of acute infections has shown that there is always present an interstitial gastritis with proliferation of interstitial connective tissue. Cases of acute gastritis, associated with fever and running a protracted course, frequently have to be differentiated from enteric fever by means of a blood culture or a serum reaction. Gallstone colic is readily ruled out by the absence of severe jaundice and pain of lesser severity.

Parasitic (or Infectious) Gastritis.—This may occur secondarily to typhoid fever, diphtheria, pneumonia, pyemia, and smallpox. The diphtheria and anthrax bacillus, the favus, thrush, and yeast fungus have been found in the stomach. Intestinal parasites in rare cases may reach the stomach. The symptoms are those of a prolonged gastritis with fever. Diagnosis may often be made only by the discovery of the causative factor in the lavage water or sometimes only at autopsy.

Phlegmonous Gastritis (Gastric Abscess—Acute Suppurative Gastritis).—This is a very rare condition characterized by a diffuse phlegmonous infiltration or a distinct abscess of the gastric wall. Only about one hundred cases have been reported. It is characterized by intense epigastric pain, high fever, severe general reaction, and prostration. Diagnosis is usually not made before death unless pus, blood, and a large number of bacteria are found in the vomitus.

Toxic Gastritis.—This form of gastritis is the result of swallowing irritating or corrosive poisons (acids, alkalies). Intense burning pain is a cardinal symptom. It is present in the mouth, under the sternum, and in the epigastrium. There is an excessive flow of saliva and extreme thirst. Collapse soon develops. Diagnosis of the condition is aided by the history, the chemical examination of the urine and vomitus, and the general characteristics of the poison, as the odor of phenol on the breath.

Chronic Gastritis.—Chronic catarrhal gastritis occurs primarily as a sequence of attacks of acute gastritis or as a result of prolonged irritation from alcohol, highly seasoned or poorly cooked and improperly masticated food. It may also be the result of a secondary local gastric disease, *e. g.*, ulcer, or of an acute or chronic general constitutional disease.

Symptoms.—The local symptoms are those of chronic indigestion. The tongue is coated, the appetite is diminished or capricious, and there is a craving for fluid, particularly acid drinks, with the meals. After eating there is a sense of fulness and oppression in the epigastrium. Gaseous eructations and pyrosis occur frequently, while nausea and vomiting appear characteristically in the morning on arising or a short time after eating. Intestinal symptoms of constipation alternating with diarrhea, rumbling (borborygmus), and vague colicky pains disturb the patient. General symptoms are pronounced. The patient is nervous, irritable, and morose. Headache, disturbed sleep, vertigo, and languor are persistent complaints.

Gastric Analysis.—The presence of lactic and butyric acid render the vomitus sour and acrid. The gastric contents show constantly an excessive amount of mucus, occurring in shreds or flakes. The test meal is poorly digested. A subacidity is present, often progressing to actual achylia, the so-called organic achylia gastrica, considered to be due to a transformation of the gastric epithelium into a type of intestinal epithelium, with lessened secretory power. Hyperacidity is rarely present, and only in the early stages. Pepsin and rennin ferment are also diminished. The motor power of the stomach is only slightly modified. Gastric inflation will show a moderately dilated stomach.

Diagnosis.—Chronic gastritis may be differentiated from a gastric neurosis, cancer of the stomach, and gastric ulcer by the following diagnostic features: (1) Long duration; (2) persistence of local symptoms; (3) occurrence of symptoms during digestive period and dependence upon quantity and quality of food; (4) absence of cachexia; (5) moderate pain; (6) absence of hemorrhage; (7) excessive flatulence; (8) excessive mucus in gastric contents.

Dilatation of the Stomach (Gastrectasia).—Gastrectasia may be acute or chronic. The acute form (gastric ileus) occurs most frequently after ether narcosis, in the course of acute infectious diseases, and after dietetic excesses. It is the result either of gastric paralysis or,

less probably, pressure by the root of the mesentery upon the duodenum. There is usually a preëxisting gastritis. The symptoms are excessive vomiting, the vomitus being bile-stained but never fecal; pronounced pressure and pain in the upper abdomen; intense thirst; rapidly developing, severe prostration going on to shock. Examination shows a distinct visible tumor, often of enormous size, in the epigastrium. The physical findings are those of an exaggerated chronic dilatation, with increased amount of clear fluid in the stomach. The diagnosis is verified by lavage and the frequent subsequent subsidence of the tumor.

Chronic Dilatation.—Two forms are recognized: the mild *atonic*, the result of wasting disease, nervous depressive conditions, the so-called *myasthenia gastrica*, and the severe *obstructive*, a sequel of pathological changes at the pyloric ring. The condition develops slowly. Marked dyspepsia is present, with excessive flatulence and pyrosis. Vomiting is frequent, and there is an excessive amount of vomitus. Dull, heavy pain is complained of, and the patient suffers from the nervous symptoms of a chronic gastritis. Tetany, varying from a mild twitching of the muscles to severe convulsions, may develop during the course of the condition. The stomach contents always show the presence of retained food as a result of the motor insufficiency. The quantity is markedly increased and is usually foul and malodorous. Free HCl is usually diminished, although in pyloric obstruction frequently much increased. After standing the stomach contents separate into layers, the upper one frothy, the middle clear, and the lower semisolid. The degree and extent of the dilatation can be readily determined by auscultatory inflation of the stomach when the gastric contents are removed.

Physical Examination.—The abdominal walls are thin and the outlines of the stomach are frequently seen. Visible peristalsis from left to right can often be noted. Palpating with one hand on the stomach and tapping with the other demonstrates a splashing sensation caused by fluid in the organ (clapotage).

The percussion note over the stomach is tympanitic. If the patient drinks a glass of water, dulness is present between the stomach and intestinal tympany, when erect, and disappears when recumbent. There is upward displacement of the apex-beat of the heart, and partial obliteration of the splenic dulness. Succussion is elicited by auscultation.

Diagnosis.—The characteristic lavage findings will clear up any question of diagnosis. The x-ray examination of the stomach after a bismuth meal will verify the diagnosis. The important point is to determine if an atonic or obstructive dilatation is present. In pyloric obstruction there is often a history of gastric ulcer or a small tumor may be felt at the pylorus. The vomiting is forceful and pain is persistent, though worse at night. Peristaltic waves are faintly seen. In the atonic form there is a lessened degree of dilatation and subjective symptoms are usually not so marked and the constitutional results less pronounced.

Stenosis of the Pylorus.—Obstruction of the pylorus is usually caused by malignant disease. It may be a result of spasm of the pylorus or of organic or functional disease, or it may result from pressure or constriction of the pylorus by some abnormal external factor, as adhesions. The symptoms and signs are those of gastrectasia from pyloric obstruction.

Congenital spastic hypertrophy of the pylorus has for its characteristic features, persistent vomiting, obstinate constipation, a tumor in the pyloric region, and rapidly developing marasmus during the first few weeks of life. In rare cases the condition may not develop until adult life.

Ulcer of the Stomach and Duodenum.—Ulcer of the stomach or duodenum occurs commonly in early and middle adult life. Ulcers are probably the result of alterations of the mucous membrane acted upon by the gastric juice, the result of trauma, embolism or thrombosis particularly in arteriosclerotic individuals, or septic infections. The relative occurrence of gastric and duodenal ulcer varies according to different authorities from 40 to 1 to 2 to 3.

Symptoms.—These are variable. The cases have been divided by Welch into four classes: those in which there are (1) no symptoms, the ulcer being found postmortem; (2) no symptoms until the sudden appearance of hemorrhage or perforation; (3) symptoms of gastralgia and of chronic gastritis; (4) typical symptoms of pain, vomiting, and hemorrhage.

Pain.—The pain is localized in the epigastrium. If severe it may be referred toward the back and up the chest. It is burning or gnawing in character, relieved at times by vomiting or the ingestion of albuminous foods, exaggerated by pressure. It occurs immediately after eating irritating food or from two to four hours later at the height of gastric digestion. In general the longer after eating the intense pain occurs, the nearer the ulcer is to the pylorus. In the intervals between the severe pain there is usually a dull, constant ache. *Vomiting* usually occurs shortly after the ingestion of food. *Hemorrhage* may be bright red and copious or may be small in amount, dark brown, and resembling coffee-grounds. The blood may be passed into the intestines in quantities sufficient to give a tarry discoloration to the stools or it may only be discovered by one of the various tests for occult blood. The string test of Einhorn will frequently show the presence of duodenal hemorrhage. One end of a white string attached to a shot is swallowed at night, the other end pinned to the night-garment. The shot is pulled up the next morning, and if bleeding has occurred, splotches of red blood can be seen on the string; where it is discolored yellow from bile if the shot has entered the duodenum.

Gastrostaxis is a term used to describe a clinical syndrome of pain, vomiting, and hemorrhage without conclusive evidence of ulcer formation. In all probability it is the result of unrecognized and undemonstrated ulcer.

Other Symptoms.—Thirst and hunger are usually pronounced. Constipation is the rule. Belching and pyrosis are frequent. The general condition commonly remains good through chloro-anemia; weakness and emaciation may result.

Physical Examination.—Two points of tenderness are the only signs demonstrated by an examination of the patient: (1) epigastric; (2) dorsal. The epigastric tenderness is usually situated in the midline just below the xiphoid. The dorsal tenderness occurs 2 or 3 cm. to the left of the spinous process of the tenth to the twelfth thoracic vertebrae notably the latter.

Gastric Contents.—In the acute forms there is usually a slight hyperchlorhydria and no change in the motor power of the stomach. In the chronic form there is insufficiency of motor power and in a majority of cases a hyperacidity. Blood is usually present macroscopically or microscopically. At times only occult blood is found by chemical examination.

Diagnosis.—The diagnostic features are (1) age; (2) gaunt appearance of the features; (3) characteristic pain and tenderness; (4) vomiting; (5) hemorrhage, gross or occult; (6) absence of marked nervous symptoms; (7) slight diminution in motor power; (8) painful pyloric spasm; (9) hyperacidity of gastric juice.

The symptom-complex of gastric ulcer and duodenal ulcer have only a few minor differences. In duodenal ulcer the pain occurs later, from three to four hours after eating, or in the early hours of the morning and is less influenced by the character of food at time of ingestion. It is situated more to the right and dorsal tenderness is more likely to be absent. The vomitus is usually, first food, then bile, and then blood. Melena is more frequent; and there is less motor insufficiency.

The Accidents (Sequelæ) of Ulcer of the Stomach.—(1) Perforation; sudden severe agonizing pain with shock, rapidly followed by peritonitis; (2) hemorrhage, which may result in death, gives the characteristic signs of internal bleeding if vomiting of blood does not occur; (3) cicatricial contractions which may cause stenosis of the pylorus or hour-glass or other contractions of the stomach wall.

Cancer of the Stomach.—Cancer occurs three times more frequently in the stomach than in any other organ.¹ It is frequently insidious in onset and difficult to diagnose until the late stages, when the possibilities of securing relief of the condition are past. Therefore the early recognition of the disease is of extreme importance. The only etiological factor which aids the diagnosis is the frequent development of cancer upon a gastric ulcer.

Symptoms of Onset.—The onset may be acute, subacute, or chronic. In the acute form the onset is ushered in by the appearance of pain in individuals free from indigestion. The pain is at first variable, it is usually relieved by food, occurs several hours after eating, and is located in the epigastrium. As the condition progresses the pain becomes

¹ Vital Statistics, 1910.

continuous, exaggerated by eating, and is not relieved by food. Subsequently the patient develops indigestion and later symptoms of gastric cancer.

The subacute form is characterized by the appearance of apparently causeless indigestion, in a person beyond middle life previously free from indigestion. Vomiting, anorexia, and gaseous eructations are frequently early manifestations of the condition.

In the chronic form the patient has suffered for years from indigestion. No changes occur in the local symptoms, although they might be slightly emphasized, so that as a rule the possibility of carcinoma is not considered until emaciation and cachexia develop.

General Symptoms.—Anorexia is present and is usually noted early. The patients frequently have an aversion to meat. Vomiting is of common occurrence. The vomitus is similar to that of chronic gastritis, later it becomes blood-streaked or dark in color (coffee-ground). The vomiting occurs after eating, and as a rule the nearer the cancer is to the cardia, the closer the time of vomiting is to the time of eating. Pain in the disease is continuous and gnawing and boring in character. Slight fluctuations in temperature are usually found. The bowels are costive, although diarrhea alone or diarrhea alternating with constipation may be present. Occult or gross blood in the feces is a finding of importance and should be sought for repeatedly if cancer is suspected.

Physical Examination.—Early, nothing is made out by examination. Later a tumor may be palpated, usually in the epigastrium. As a result of metastasis the liver becomes enlarged and nodular. Glandular enlargement may be noted in the supraclavicular, axillary, and inguinal glands. Boas has called attention to the frequency of metastasis to the rectum, demonstrated readily by rectal examination.

The patient presents the objective symptom of cachexia. He is emaciated and anemic. The face is often of a yellowish and straw-colored hue, suggesting jaundice except, for the pallor of the sclera. The skin is flabby and boils are common. Subcutaneous hemorrhages may be noted. There is slight edema of the ankles.

Examination of the Stomach Contents.—Free hydrochloric acid is absent or diminished. It must not be forgotten, however, that in rare cases it is increased. Organic acids, lactic and butyric, are found when there is much fermentation. Evidence of motor insufficiency of the stomach is found if systematic examinations of the motor power of the stomach are made, and it will be found to rapidly increase.

Microscopically.—If cancerous fragments are found the diagnosis is assured. Erythrocytes, many pus cells, and much epithelium are extremely suggestive findings. The Oppler-Boas bacillus is frequently discoverable.

Urine.—Indican is increased.

Blood.—There is an absence of digestive leukocytosis. A marked anemia is present in the last stages, but not as severe as in pernicious anemia.

Diagnosis.—The diagnosis of gastric cancer must be supplemented in questionable cases, by the x-ray examination and occasionally the gastroscope. Certain so-called specific tests for gastric cancer have been elaborated, but are not to be recommended as they are inconstant and unreliable.

The correct diagnosis is based on the careful study of the symptoms bearing in mind the age of the patient, the frequent occurrence of pain, the association of vomiting with eating of food, the results of metastasis and the rapid course of the condition. Valuable adjuvants are the absence of free hydrochloric acid in the gastric contents, the presence of occult blood in the feces, the rapidly increasing motor insufficiency of the stomach, and the fluoroscopic examinations.

Syphilis of the Stomach.—Four gastric lesions may occur as a result of syphilis; all are very rare: (1) ulcer; (2) gumma; (3) pyloric stenosis (4) diffuse cellular infiltration.

The diagnosis can be made only by the specific serological test and the effect of treatment.

Tuberculosis of the Stomach.—Gastric tuberculosis occurs as ulcerations of the stomach wall. There are no distinct symptoms of the condition, the diagnosis being based on the chronicity of the symptoms, the failure of treatment, the presence of tuberculosis elsewhere, and the constitutional reactions of tuberculosis.

Volvulus of the Stomach.—This is a rotation of the organ upon its long axis as a result of malposition. The symptoms are pain radiating toward the back, acute gastric meteorism, hiccough, and the impossibility of passing the stomach-tube.

Functional Disorders of the Stomach.—Gastric Neuroses.—These are characterized by changes in the secretory, sensory or motor functions of the stomach without demonstrable pathological changes.

The following modified table of Ewald is a classification of the various neuroses midway between the symptomatic and etiological:

I. CONDITIONS OF IRRITATION

<i>A. Sensory</i>	<i>B. Secretory</i>	<i>C. Motor</i>
Hyperesthesia	Hyperacidity	Cardiospasm
Nausea	(Hyperchlorhydria)	Pylorospasm
Anorexia	Hypersecretion	Hypermotility
Parorexia	(Gastrosuccorhea)	Eruetation
Bulimia	Gastroxynsis	Regurgitation
Polyphagia		Vomiting
Akorea		Tormina ventriculi
Sitophobia		Merycism
Gastralgia		Pneumatosis
		Singultus

II. CONDITIONS OF DEPRESSION

Anesthesia	Hypacidity	Atony
	Anacidity	Pyloric insufficiency
	Gastromyxorrhea	Cardiac insufficiency

III. MIXED FORMS

Dyspepsia nervosa

It should be remembered that in the above classification the symptoms may occur singly or in combination and may be a manifestation of organic disease as well as functional disturbance of the stomach, or may arise reflexly from disease of other organs.

Etiology.—To discuss the various etiological factors concerned in the production of gastric neuroses is beyond the scope of this book. In general it may be said that they occur most frequently in midlife, in persons of peculiar nervous temperament and as a result of faults in the habits of life. It is important to bear in mind the fact that so-called gastric neuroses are frequently the result of some primary condition, either in the stomach or elsewhere in the economy and that a diagnosis of a gastric neurosis should not be made without a most thorough investigation.

General Symptoms of Gastric Neuroses.—The patient is neurasthenic. He complains of various kinds of headache and neuralgic pains, of vertigo, insomnia and other nervous symptoms. Constipation is the rule, although diarrhea or constipation alternating with diarrhea may be present.

I. (a) SENSORY NEUROSES OF IRRITATION.—*Hyperesthesia.*—This may be mild, continuous pain, aggravated by irritating foods, or it may occur simply as a feeling of discomfort, or gnawing in the stomach. It may be a manifestation of functional disorder of the stomach or may be found in chronic gastritis, hysteria, cerebral tumors, and meningeal irritation. While the test meal is usually normal, if organic disease is present the hyperesthesia is usually a result of hyperacidity.

Perversions of the Appetite.—*Boulimia* or hyperorexia nervosa is a temporary or permanent feeling of hunger. *Anorexia* is a loss of appetite or repugnance for food. *Parorexia* is a perversion of appetite, manifested by a desire for unusual foods. *Akoria* means the absence of feeling of satiety after eating. *Sitophobia* is the fear of food. *Polyphagia* is the condition in which excessive amounts of food are required to satisfy the feeling of hunger.

Nausea.—The nausea may be continuous or intermittent, usually coming on at first independently of digestion. It is always associated with anorexia.

Gastralgia.—The pain may be a result of peripheral or central sensory stimulation of the vagus. It is sudden, agonizing, referred to the epigastrium, and usually without regularity in time of appearance. It is relieved some times by eating and pressure upon the epigastrium. The attack is attended by more or less collapse. The gastralgia due to disease of the central nervous system, *e. g.*, the gastric crises of tabes, are often most puzzling. The pains begin in the groin suddenly and ascend to the epigastrium, where they remain localized. With the pain there is uninterrupted and painful vomiting which may continue for hours. A subnormal temperature is found and other signs of shock are present. *Neurasthenic gastralgia*, characterized by a boring sensation or a feeling of fulness and weight in the epigastrium, occurs most

frequently in neurasthenic females at the time of the menstrual period. *Hysterical gastralgia* is diagnosed by the presence of the subjective and objective symptoms of hysteria.

Gastralgias are differentiated from the pains of organic disease of the stomach by the character of the secretions, which are usually normal, though at times with increased amount of hydrochloric acid by normal motor sufficiency; by the absence of melena; by the presence of other neurasthenic symptoms; by the loss of the knee-jerks in tabetic gastralgia; and by the characteristic sudden causeless onset.

The diagnosis between gastralgia and nephritic or gall-stone colic is made by noting the location and radiation of the pain. In renal colic the pain is lower down and radiates toward the genitalia. In gall-stone colic it is located farther to the right, referred to the right side of the body, and is most often accompanied by jaundice.

(b) SECRETORY NEUROSES OF IRRITATION.—*Hyperacidity* or *hyperchlorhydria* is a frequent condition. Most frequently due to organic disease or to reflex irritation of the stomach by gall-stones, chronic appendicitis and allied conditions, it may at times be a pure functional neurosis often accompanying melancholia, neurasthenia, or hysteria. The symptoms are heart-burn with acid eructations, burning, boring epigastric pain relieved by albuminous food, and constipation. Examination shows some epigastric tenderness.

The gastric contents after a test meal, contain an increased amount of hydrochloric acid with corresponding increase in the total acidity which may possibly reach 120. There is an increase in the quantity of the filtrate; about 80 c.c. is the usual amount found. Starch digestion is poor while protein digestion is good. There is no evidence of impairment of motor function of the stomach.

Hypersecretion and gastrosuccorrea occurs in two forms, the periodic and the chronic or continuous (Reichmann's disease). In the periodic form, a pure neurosis, there takes place a sudden accumulation of a large amount of fluid in the stomach accompanied by gnawing pain, pyloric spasm and at times vomiting of large amounts of watery gastric secretion. It may last several days. The fasting gastric contents are excessive, the filtrate yielding over 100 c.c. of fluid. There is also an accompanying hyperacidity but no evidence of motor insufficiency. In the chronic form there is probably either actual pathological changes in the mucosa to account for the continuous excessive secretion, or the condition may occur secondarily to reflex irritation from pathological conditions elsewhere, as from gall-stones, chronic appendicitis, and similar disorders. The symptoms and findings are similar to those of the periodic form except that they are persistent. In addition there usually develops sooner or later a dilatation of the stomach with motor insufficiency.

Digestive gastrosuccorrea is a term applied to an excessive amount of fluid occurring after digestion. The filtrate runs up to 100 c.c., and there is usually a normal acidity.

Gastroxynsis is a form of periodic gastrosuccorhea accompanied by severe headache, much like migraine in character.

(c) *Motor Neuroses of Irritation*.—*Cardiospasm* is a condition in which there is a spasmodic contraction of the cardia. The symptoms make themselves manifest during digestion by a sudden feeling of constriction behind the lower sternum. Dysphagia is present in the effort to force food into the stomach. If this cannot be done, then there is a regurgitation of indigested food. It may be differentiated from stricture of the esophagus by the passage of bougies or sounds which proceed to the cardia and then meet an obstruction, which readily gives away and which gradually becomes obliterated as the instrument is passed to and fro through the cardiac orifice. If the condition progresses to dilatation of the esophagus the use of the esophagoscope may be necessary to make a diagnosis.

Pylorospasm is rare as a neurotic manifestation; common as a secondary manifestation of other disorders as hyperacidity, hypersecretion, hyperesthesia, ulcer, gastric ptosis, pyloric cancer and reflex irritation from disease of other abdominal organs. The condition is characterized by the occurrence of sudden severe epigastric pain at the height of digestion, accompanied by eructation of gas and at times vomiting of food. As the condition becomes more aggravated the symptoms of gastrectasia develop. Examination of a patient during an attack discloses some localized tenderness, and perhaps the firm, hard pylorus can be palpated. It is differentiated from organic stricture by the thread test, the shot passing eventually into the duodenum and the string becoming bile-stained.

Hypermotility is diagnosticated if there is an absence of gastric secretion when lavage is performed for the removal of a test meal.

Eructations may be the result of motor insufficiency or may come from the swallowing of air. The gas is odorless and inoffensive.

Regurgitation is a neurotic condition in which food is brought up into the mouth and then expelled. *Merycism* is a similar condition except that the food is chewed and then reswallowed. The conditions are differentiated from vomiting by the absence of nausea.

Vomiting may be a direct neurosis affecting the vagus centre or may be reflex from diseases of the eye, nose, stomach, kidneys, in fact from any organ of the body. *Singultus*, hiccough, a sudden contraction of the diaphragm occurs occasionally as a manifestation of a gastric neurosis. *Pneumatosis* is an excess of air in the stomach, frequently found in dilatation of the stomach. *Tormina ventriculi* (peristaltic unrest) is characterized by gurgling and borborygmi. Examination will show at times visible peristaltic waves, although pyloric obstruction cannot be demonstrated.

II. (a) SENSORY CONDITIONS OF DEPRESSION.—Anesthesia occurs in conditions of depression, manifested by perversions of appetite in some form.

(b) SECRETORY NEUROSES OF DEPRESSION.—*Hypacidity, Anacidity, or Achylia Gastrica*.—This condition is characterized by diminution or total absence of free hydrochloric acid. It occurs (1) organic diseases of the stomach, as cancer and long-continued chronic gastritis; (2) other diseases as diabetes, acute infections, chronic wasting diseases, and blood diseases, as chlorosis and pernicious anemia; in pernicious anemia there is an actual atrophy of the secretory structure of the stomach; (3) functional disturbances of the stomach in neurotic individuals.

The symptoms of the condition are not characteristic. There is irregular vomiting, gaseous eructations, anorexia, and vague distress. Diarrhea is the rule. The condition is to be differentiated from chronic gastritis, pernicious anemia, and cancer of the stomach.

Gastromyorrhea is a neurosis of secretion characterized by excessive mucus (over 25 c.c.) in the gastric contents after a test meal. Two forms are recognized. A rare intermittent and a more frequent continuous form. There are no characteristic symptoms of the condition. Continuous gastromyorrhea usually is accidentally discovered in the procedure of lavage. The intermittent form is characterized by paroxysms of severe vomiting, the vomitus being composed almost entirely of mucus.

(c) MOTOR NEUROSES OF DEPRESSION.—*Atony* or motor insufficiency of the gastric musculature depends very largely upon the mental condition of the individual. Cannon has shown experimentally that in a cat abnormal mental states are accompanied by cessation of the movements of the stomach. Acute mental upsets therefore cause an absence of motility of the stomach. Worry, fatigue, anxiety, all have a like power, in all probability, of disturbing motility and secretion, causing gastric neuroses. It can be seen, therefore, that as a result of these mental or neurotic conditions atony may develop. It gives no symptoms early, but sooner or later symptoms of a mild, first-degree gastrectasia develop. These may subside or become progressively worse, eventually terminating in a dilatation of the stomach of high degree.

The gastric analysis will demonstrate the decreased motility. (See test for Gastric Motility, page 390.)

Pyloric and cardiac insufficiency are manifested by diarrhea in the first condition and regurgitation of food in the second.

III. MIXED NEUROSES.—NERVOUS DYSPEPSIA.—In the neuroses of this character the functions of the stomach are normal, but the patient suffers from one or more or several of the manifestations of functional disturbance of the stomach. The one characteristic feature is the mildness of the symptoms of gastric and intestinal disturbance. Associated with the local symptoms are the nervous symptoms of the condition recognized as neurasthenia when no organic lesion can be found. The diagnosis is based upon the general symptomatology of neurasthenia, the variable and inconstant gastric complaints and the failure to demonstrate organic disease of the stomach.

DISEASES OF THE INTESTINES

The intestine is a canal the function of which is to propel the partially digested food discharged from the stomach and to digest and absorb it. Alterations of the function of the intestine as a canal gives rise to distinctive symptoms. Either its movements are too frequent and rapid, causing diarrhea, or too sluggish, causing constipation. Alterations of the intestinal canal as an organ of digestion and absorption are manifested locally by flatulence, at times pain, changes in the character of the stools and diarrhea; generally, by impaired nutrition. The intestines also have the function of excretion, as certain toxins, drugs, and poisons are eliminated through the intestine.

Intestinal Indigestion.—Intestinal indigestion is a term applied to the symptoms resulting from functional derangements of the intestine without actual histological changes. These may be due to neuroses of sensation, secretion or movement, to diminution of the bile, pancreatic and intestinal secretions, or to excessive bacterial action.

Acute intestinal indigestion, the result of irritative food and dietetic errors, is attended with colic, flatulence, and diarrhea. There are accompanying symptoms of gastritis, as nausea and some pain in the upper abdomen. The feces may be clay-colored or unchanged.

Chronic intestinal indigestion, the result of frequently repeated acute attacks or prolongation of an acute attack, very rapidly develops into a catarrhal enteritis, with actual pathological change in the intestinal walls and with symptoms of that condition.

Acute Enteritis.—1. **Acute Catarrhal Enteritis.**—This results from ingestion of irritating food or of toxic food substances, from the action of certain drugs (arsenic, antimony, and mercury), from extension of inflammation from neighboring structures, and occurs in the course of chronic cachectic diseases, in diseases of the heart and as a result of acute infectious diseases.

Symptoms.—Diarrhea is the chief symptom. The stools varying in frequency and in color. Colicky pains about the umbilicus, with borborygmus and frequent desire to stool are usually present. Fever of the remittent type is always present and there is more or less debility. The physical signs show some distention of the abdomen, diffuse tenderness, and tympany on percussion. A leukocytosis may be noted.

Diagnosis.—The diagnosis is simple; the difficulty lies in determining the cause of the diarrhea. An attack occurring as a result of irritating food can be told by the character of the feces. If the diarrhea is caused by poisons or drugs, other symptoms of poisoning usually present themselves. In arsenic and antimony poisoning there is always vomiting, extreme diarrhea and rapid collapse. Catarrhal enteritis due to acute infectious disease is attended by symptoms of the respective affections. If the diarrhea occurs in the course of tuberculosis or syphilis it is the result of a secondary infection of the intestines.

Diagnosis of Part Most Affected.—The whole gut usually partakes of the inflammation which, however, may manifest itself with greater vigor in certain parts. Thus if the duodenum is the part chiefly affected, there are usually pronounced gastric symptoms associated with jaundice and but little diarrhea. If the remaining small intestine is chiefly involved, gastric symptoms are less marked, the stools are mixed with mucus, there is greater rumbling and less tenesmus than when the larger bowel is more affected. In the large intestine the inflammation causes the discharge of large amounts of mucus not intimately mixed with the stools. There is tenderness along the course of the colon and some tenesmus. Acute proctitis gives rise to tenesmus, to small, frequent stools, pain and tenderness in the left lower quadrant of abdomen, and tenderness about the anus.

2. **Phlegmonous Enteritis.**—This is extremely rare. There is present extreme pain and collapse, rapidly going on to death.

3. **Diphtheritic Enteritis.**—This is a variety of secondary inflammation of the bowel characterized by the formation of a false membrane. The condition may be symptomless or diarrhea and abdominal pain may be present, but there is nothing characteristic of the condition except the passage of portions of the membrane.

4. **Cholera Morbus (Cholera Nostras).**—The attack is characterized by the sudden onset of severe vomiting and intense abdominal pain, paroxysmal in type. This is soon followed by fecal diarrhea which later becomes profuse and watery. Severe general manifestations of collapse and extreme prostration are usually present. The attack subsides in twelve to twenty-four hours or is followed by gastro-intestinal catarrh.

5. **Enterocolitis or Inflammatory Diarrhea.**—This occurs in young children and is characterized by abruptness of onset, rapid exhaustion, abdominal pain, high fever, and diarrhea. The movements are greenish in color and extremely offensive.

6. **Cholera Infantum.**—This is a particularly severe form of diarrhea in babies which results from the taking of infected milk or food. The onset is abrupt with severe vomiting and purging. The course of the disease is so rapid that in a comparatively short time stupor and coma appear, which in 90 per cent. of cases, go on to death.

Chronic Enteritis.—Chronic catarrhal enteritis may follow an acute attack or occur secondarily to gastric achylia, to portal congestion the result of diseases of the liver or chronic heart disease, or may be due to intestinal parasites. Epidemic (tropical) dysentery and amebic dysentery are discussed elsewhere.

Symptoms.—The chief symptom is constipation alternating with diarrhea or simply diarrhea. The stools contain undigested food, sometimes pus, blood, and mucus. The feces may show parasites or their eggs. Local abdominal symptoms of rumbling, tormina, flatulence are present. Chronic catarrhal gastritis with its train of symptoms is a constant companion of chronic enteritis. The chr

inflammatory process of the two organs results in pronounced neurasthenia, considerable anemia, and emaciation.

Parasites.—Parasites, their ova or spores, enter the intestinal tract with food or water. They may be vegetable organisms as bacteria or fungi, or animal organisms as forms of protozoa or vermes. Certain vegetable organisms (bacteria) are always present normally in the intestines, while abnormal bacteria as a rule cause no symptoms as long as the intestinal mucous membrane is intact. The symptoms produced by the protozoa are those of local inflammation plus a general toxic influence or a disturbance of assimilation. Thus *Amœba coli* cause intense bloody mucus diarrhea with great tenesmus and general emaciation.

Vermes may be symptomless or else cause symptoms of a general nature, notably anemia, neurasthenia, and convulsions, or local symptoms of catarrhal inflammation with its varied symptoms.

The several forms of *tape-worm* (*Tœnia solium*, *Tœnia saginata*, *Bothriocephalus latus*, etc.) do not cause local symptoms but more frequently cause headache, lassitude, weakness; in children, convulsions and restlessness at night.

The *round-worm* (*Ascaris lubricoides* and *Oxyuris vermiculrris*) cause symptoms chiefly of a gastro-intestinal enteritis. The oxyuris also causes annoying symptoms of itching and burning at the anus with tenesmus. The *strongyloides* produce local intestinal symptoms and general symptoms of severe anemia.

Uncinaria americana (hook-worm) invading the organism through the skin or by the mouth, attacks chiefly the upper alimentary tract. They pierce the mucous membrane to suck blood from the capillaries. The tiny wounds they make continue to bleed after they leave, as a result of their secretion which prevents the clotting of the blood. These numerous small hemorrhages soon produce an anemia, varying from slight to a very severe grade, according to the resistance of the individual and the number of parasites. There develops from the anemia typical symptoms of lassitude, loss of strength and general weakness. In certain localities, notably in Southern States, primary anemia should never be diagnosticated without a thorough examination of the stools.

Appendicitis.—This is the most important affection of the intestinal tract. It is distinctly a disease of early life and occurs more frequently in males than in females. Anatomically, appendicitis is probably the result of some condition which results in the retention of intestinal contents within the appendix with secondary multiplication of bacteria and with probably an increase in their virulency. It is customary to subdivide acute appendicitis into various forms as catarrhal, interstitial, ulcerative or gangrenous. Clinically, this is practically impossible. A slight inflammatory condition of the appendix may set up a train of pronounced and severe symptoms. Conversely a gangrenous appendix may cause only mild and innocuous symptoms. Therefore.

symptoms will be merely divided into those occurring without perforation and those which present the symptoms of a perforated appendix.

Appendicitis without Perforation.—The attack is usually ushered in by *pain*. This is diffuse at first but soon becomes localized in the right lower quadrant of the abdomen. Later it may be referred to the umbilicus. At times, if the appendix is retrocecal, the pain may be in the back or below the liver. If the appendix dips down into the pelvis the pain may be on the left side of the abdomen and associated with vesical irritability. The pain varies from a dull ache to a severe colic-like pain. Nausea and frequently vomiting attend the severe pain. There is usually an early concomitant diarrhea, succeeded by constipation and resulting abdominal distention. Firm pressure over the seat of the pain elicits *tenderness*. This is more marked and more severe over McBurney's point, a circumscribed spot midway between the umbilicus and the anterior superior spine of the ileum. Attempts to palpate the appendix are unwarranted and dangerous. *Rigidity* of the right rectus and overlying muscles is another sign of extreme importance. *Fever* is an inconstant symptom, though always present at some time or another in the course of the disease.

A *leukocytosis* of over 15,000 is usually found. Associated with a normal differential count a leukocytosis shows a moderate infection with good power of resistance in the patient. The higher the polynuclear neutrophiles go, the more severe is the infection. A low leukocytic count with marked increase in the neutrophiles (over 90 per cent.) indicates a severe infection with poor resistance.

As said before it is impossible in most cases to tell from the clinical picture the extent and severity of the inflammatory involvement. In general, the more pronounced the local symptoms are the more severe is the infection. Marked constitutional symptoms, out of proportion to the local signs, frequently denotes a necrotic or gangrenous appendix. Increased and spreading local signs indicate a local peritoneal involvement becoming general.

Appendicitis with Localized Abscess.—Before perforation occurs the patient may have had an apparent mild attack of appendicitis for a few days; or the attack may have lasted for a long time without any manifestation except colicky pains. This sequence of events may result in the formation of an abscess, if adhesions wall in the inflamed appendix. With the formation of the abscess the general symptoms undergo a change and symptoms of suppuration become pronounced. There are chills, fever, sweats, loss of appetite, and general prostration. The early rigidity gives way to a boggy sensation with edema of the overlying skin. Some dulness is present on percussion over the appendix. Examination per rectum may disclose a fluctuating mass in the rect fossa. If operative procedures are not resorted to the abscess may rupture or symptoms of septicemia develop.

Appendicitis with Perforation.—Perforation occurring in the course of an acute attack may manifest itself by sudden sharp pain or sud-

increased local tenderness. It may be shown only by a quick rise in temperature with rigors and chills; if a very severe infection, a fall of temperature, and general signs of shock. Soon the patient presents all the signs of a general peritonitis; diffuse board-like rigidity of the abdominal muscles, increasing pulse-rate, and severe prostration.

Differential Diagnosis.—The cardinal symptoms of appendicitis, pain, tenderness, and rigidity, suffice to make a diagnosis in a typical case. The atypical forms, however, frequently present a series of symptoms which necessitate a differentiation from other acute abdominal affections and from a few conditions outside the abdomen, which in turn often present symptoms most suggestive of appendicitis.

Typhoid Fever.—Occasionally the onset of typhoid fever may simulate appendicitis. The history of malaise for a few days preceding the pain, the milder local symptoms, and the leukopenia will aid in differentiating the condition. Rarely appendicitis develops as a result of the typhoid ulceration in the cecum.

Pneumonia.—A right-sided pneumonia or pleurisy or a pericarditis may be manifested early only by signs and symptoms characteristic of appendicitis. Careful examination will result in a proper diagnosis, but the possibility should never be forgotten, as frequently a diagnosis of appendicitis is made without a proper examination of the thorax.

Malaria occasionally begins with acute abdominal symptoms and may be suspected only by the discovery of an enlarged spleen. A blood examination will show the true condition.

Renal Colic.—When the appendix is pointed downward it may give symptoms comparable with those occurring in renal colic. The pain in the latter condition is sharp, cutting, and more severe. Disturbance of micturition is more pronounced, and the urine usually shows blood cells grossly or microscopically.

Diehl's crises in floating kidney are differentiated by the absence of fever and by the discovery of the swollen palpable kidney.

Gastro-intestinal colic is at times mistaken for appendicitis. The history of dietetic errors, the absence of localized pain with tenderness, and the afebrile course readily distinguish the former from the latter condition.

Acute colitis due to stercoral accumulations is difficult and at times impossible to diagnosticate. It is usually less acute in onset and the intestinal symptoms are more pronounced.

Acute Intestinal Obstruction.—Stercoraceous vomiting, early severe constitutional symptoms, and diffuse distention will be results of the obstruction not found in appendicitis.

Inflammation of the gall-bladder and biliary colic may simulate appendicitis or may be simulated by appendicitis. Careful physical examination will show the location of the pain and tenderness in appendicitis to be lower in the abdomen. The presence of jaundice is an invaluable sign in differentiating the two conditions.

children. The symptoms are those of diarrhea and colicky pains associated with rapid emaciation and constitutional signs of tuberculosis. Examination shows a much distended tympanitic abdomen which may later contract. The mesenteric glands along the spinal column are found to be enlarged, and later, when the abdomen has retracted, the coiled-up intestines may be palpated.

Syphilis of the Intestines.—Lesions of congenital and acquired syphilis occur rarely in the intestines. The symptoms are those of simple intestinal ulceration or stricture. The diagnosis is based upon the history of the associated findings of syphilis and the positive serological reaction.

Amyloid Disease.—Amyloid disease of the intestines presents one constant symptom—frequent and watery diarrhea. The diagnosis is based upon the recognition of some cause for amyloid degeneration together with its occurrence in other organs.

Embolism and Thrombosis of the Mesenteric Arteries.—This is usually a result of atheromatous plaques of the mesenteric arteries. Sudden severe abdominal cramps, followed by vomiting and diarrhea, both of which soon become hemorrhagic and copious, rapid fall in temperature with signs of shock, tympanites, and distention of the intestines, together with free fluid in the abdominal cavity, are the characteristic symptoms of the condition.

Angina Abdominalis.—This occurs in patients with arteriosclerosis, usually after physical effort. It is characterized by paroxysmal attacks of abdominal pain followed by rapid meteorism. The absence of fever, the high blood-pressure and the rather rapid subsidence of the condition, differentiates it from other acute abdominal conditions.

Intestinal Diverticula.—These are pouchings of the intestinal wall, true diverticula, and are either congenital, as Meckel's diverticulum, or the result of congenital predisposition. False diverticula are hernial protrusions of the mucous coat through the muscular coat (Stengel). The false diverticula are found for the most part in the descending colon and sigmoid flexure. Diverticula are subject to the same acute and chronic inflammatory processes as the appendix and present much the same clinical picture when inflamed. At times the symptoms are largely obstructive in character, closely resembling those of intestinal carcinoma. Undoubtedly many obscure pains in the left lower quadrant of the abdomen are a result of a diverticulitis.

Intestinal Obstruction.—**Etiology.**—Intestinal obstruction may be acute or chronic. The pathogenic factors may be outside of the intestines, in the intestinal wall, or within the intestine.

I. *Disorders Outside of the Intestines.*—(1) Pressure of tumors; (2) constricting bands; (3) hernial orifices; (4) adynamic obstruction; (5) volvulus.

II. *Disorders of the Intestinal Wall.*—(1) Invagination or intussusception; (2) cancer or tumors; (3) strictures the result of contractions from healing ulcers; (4) congenital kinks of the intestines.

III. *Accumulations within the Intestines.*—(1) Feces; (2) foreign material, worms, food, etc.; (3) impacted gall-stones.

Acute Intestinal Obstruction.—This may develop suddenly from strangulation of the intestines in hernial orifices or by bands, or as a result of volvulus or intussusception. It is a sequel of peritonitis and frequently develops in the course of chronic obstruction.

SYMPTOMS COMMON TO ALL FORMS OF ACUTE OBSTRUCTION.—The local symptoms depend upon the nature and the seat of the obstruction. *Constipation* is the main symptom. It is so complete as a rule that not even flatus escapes. *Pain* may be at the seat of the obstruction or about the umbilicus. It is intense and colicky and associated with tenderness over the painful part. *Vomiting* occurs soon, the result of reflex irritation of the stomach and of reversed peristalsis. At first watery and inoffensive it soon becomes apparently fecal in character and later truly fecal. General symptoms are those of extreme prostration and shock. Just why this should be so pronounced and why it is not known. It results from the accumulation of extremely toxic fluid in the intestine above the obstruction. The fluid is so poisonous that after experimental intestinal obstruction its injection in normal animals will kill them.

PHYSICAL EXAMINATION.—The patient is extremely prostrated, with the general signs of collapse or shock. The abdomen is distended but soft and flaccid, with some tenderness over the site of the obstruction. Occasionally a tumor can be palpated or peristalsis may be seen above the constriction. Meteorism is discovered above the obstruction by the tympanitic note on percussion. Muffled dulness may be present over the site of the greatest tenderness.

DIFFERENTIATION OF THE VARIOUS FORMS OF ACUTE OBSTRUCTION.—*Strangulation* of the intestines by bands occurs most frequently in the ileocecal and pelvic regions. A history of a former peritonitis may be obtained from the patient, or a history of inflammation of a peritoneal covered structure, as the appendix, Fallopian tube, or gall bladder. Strangulation may be the result of incarceration of a knuckle of gut in an external hernial orifice or may result from the formation of an internal hernia, the intestine slipping into the jejunal, pericecal or intersigmoid fossa, or through the foramen of Winslow or an aperture in the omentum.

Strangulation occurs as a rule in middle life. The attack commences suddenly and usually presents the typical symptoms of obstruction. The pain is severe and paroxysmal. Local tenderness is not pronounced so early as in other forms of obstruction. If the strangulation is through an external hernial orifice, examination will disclose it.

Volvulus is a rare condition, occurring usually late in life. It is a result of twisting of the bowel upon an abnormally long mesentery. It occurs most frequently in the sigmoid flexure and hence the pain and tenderness are more frequent on the left side and often associated with rectal symptoms. The pain occurs early, is not so severe as

the other forms of obstruction, and is frequently constant with exacerbations. Vomiting is present late. Meteorism is early and pronounced.

Intussusception is an invagination of the intestines, the upper portion being pushed into the lumen of the part immediately below. It originates most frequently at the ileocecal valve, and children are particularly likely to be affected. The pain is intense and paroxysmal and early. Vomiting is not so severe and does not appear as early as it does in obstruction higher up. The characteristic diarrhea which is present is an important differential point. It occurs early in the condition and may continue. The stools at first contain fecal matter, but soon the movements become bloody and mixed with mucus. The evacuations are attended with marked tenesmus. Examination will disclose a palpable tumor, characteristically sausage-shaped. Tenderness over this tumor is marked. Meteorism is usually absent. Visible rectal examination often discloses the dark gangrenous bowel, which will be found to be tender and painful if palpated.

Adynamic obstruction from paralysis of the bowel is usually a result of peritonitis and is differentiated from mechanical obstruction by the presence of fever following an initial chill, by the history of or determination of some cause for the peritonitis, by the absence of fecal vomiting and by the early rigidity of the abdominal wall. It must be remembered that general peritonitis itself may develop in from twenty-four to forty-eight hours after the initial symptoms of acute obstruction. Obstruction from paralysis of the bowel, the result of embolism or thrombosis of the mesenteric artery is differentiated from mechanical obstruction by its occurrence in arteriosclerotic individuals with high blood-pressure and the colicky character of the passages.

Acute obstruction developing as a result of stenosis of the bowel may appear suddenly. It presents the characteristic symptoms of any acute obstruction plus the earlier symptoms of the chronic obstruction.

THE SITE OF OBSTRUCTION.—If a diagnosis of the cause of obstruction is made, the usual position of that form should be borne in mind. The site of obstruction is indicated by (1) location of the pain; (2) character of the stools; bloody and mucus stools at times if obstruction is low; (3) character of the vomiting; more severe and earlier if the obstruction is high, and often not fecal; (4) degree of meteorism; the lower the obstruction the greater the meteorism; (5) rectal examination; obstruction of the large bowel is visible through the proctoscope at times; (6) peristalsis, in thin subjects visible at times down to the obstruction; (7) changes in urine; the higher the obstruction the greater the indicanuria and likelihood of anuria; (8) general condition; the more severe and rapidly developing the symptoms the higher the obstruction.

DIFFERENTIAL DIAGNOSIS.—Acute obstruction is differentiated from various acute abdominal conditions as acute hemorrhagic pancreatitis, acute appendicitis and ruptured gastric ulcer by the pronounced meteorism, the fecal vomiting and the absolute constipation. Chronic peritonitis and tuberculous peritonitis with vomiting and severe

stipation run a longer course than acute obstruction, prostration and pain are not so severe, and fecal vomiting is absent. Marked abdominal distention in the course of acute infectious diseases or an enterocolitis do not show absolute constipation, an enema bringing away fecal matter.

Chronic Intestinal Obstruction.—Stenosis of the intestines is gradual in onset and occurs only in adults. The characteristic symptoms are constantly increasing constipation, spurious diarrhea, and irregular colicky pains. If the obstruction is high up in the small intestine constipation is usually less pronounced, while local gastric symptoms of gastrectasis, eructations, nausea, and vomiting predominate. Local conditions, indicating the cause of the obstruction, may be present, as a fibroid or ovarian tumor, a cancer of the intestinal wall, a history of a previous localized peritonitis, or some such causative factor may be obtained. General symptoms are those of inanition with the nervous train of symptoms resulting from intestinal toxemia.

Examination of the abdomen shows the presence of peristaltic waves, particularly if the obstruction is low. There may be some distention or the abdomen may be flat. In either case the abdominal walls are relaxed. A fecal tumor can be palpated when the obstruction is low; if high up the stomach will be found dilated. The stools show mucus and at times blood. Rarely there may be alterations in the shape of the stools. X-ray is frequently a valuable aid in arriving at the diagnosis.

Cancer of the Intestines.—Cancer of the intestines occurs most frequently either in the cecum, colonic flexures, or rectum. It may manifest itself only by progressive emaciation and cachexia until the sudden occurrence of obstruction of the bowel. Local symptoms depend upon the location of the carcinoma. If in the duodenum symptoms of gastric retention are marked, jaundice is infrequently present, and a palpable tumor to the right of the pylorus and less movable than a pyloric cancer may be palpated. If the remaining part of the small intestine be involved, pain of a colicky nature is a constant symptom. With this occurs symptoms of partial obstruction as pain and constipation alternating with diarrhea. Hemorrhage from the bowel is usual. It may, however, be recognized only by chemical examination of the stools. Cancer of the colon is manifested by two important symptoms, colicky pain and constipation, perhaps alternating with diarrhea. If the tumor obstructs the lower bowel, there may be alterations in the shape of the bowel movements, as ribbon-shaped stools. Blood, mucus, and pus cells are found in the stools when there is ulceration of the neoplasm. Cachexia will develop sooner or later; as a rule, the nearer the cancer is to the stomach the more rapid and pronounced the cachexia.

Examination.—The most characteristic physical finding is a tumor which is usually tender, lying in the long axis of the intestine and movable if in the small intestine, fixed if in large intestine. Its detection is facilitated by having the patient get on his hands and knees and then palpating the abdomen with the flat of the hand. Peristalt

waves can be seen at times approaching the site of the tumor. Gurgling may be heard by auscultation over a tumor, and is due to the fecal stream being forced through the narrowed lumen of the intestine. Inflation of the colon may outline the cancer if in the lower part of the intestine. The proctoscope brings into view those cancers that lie in descending colon, sigmoid flexure, and rectum. X-ray will frequently demonstrate a malignant growth in the intestine.

Diagnosis is based upon (1) the general symptoms of cancer; (2) the tumor; (3) constipation, becoming progressively worse.

Boas insists upon the importance of the sudden occurrence of moderate obstruction in healthy persons over thirty-four years of age, and considers the obstruction of peristaltic waves, particularly sudden lightning-like contractions, very important.

Benign tumors of the intestine occur in the large bowel. Hemorrhage and tenesmus are the principal manifestations.

Diseases of the Colon.—Many of the diseases of the colon are discussed under the various intestinal diseases, but as the colon may be the seat of disease which is not directly manifested elsewhere in the intestinal tract or may possess certain congenital malformations foreign to the remaining gut, these conditions will be briefly described, bearing in mind, however, that affections of the colon will probably cause symptoms of functional disturbances in the small bowel.

Movable Cecum.—This is a result of congenital malformation of the mesocolon of the cecum of such a nature that for some distance along the ascending colon it maintains the type of the mesentery and is not attached to the parietal peritoneum (Sailer). The symptoms are the result of the displacement and distention of the cecum. Vague colicky pains in the right lower abdominal quadrant, constipation, with mucus in the stools, and intestinal flatus are frequent findings. Often the symptoms are such that a chronic appendicitis is suspected and appendectomy is performed without the expected relief of the symptoms. The patients are neurasthenic and poorly nourished. Physical examination will disclose gurgling in the right iliac fossa; some tenderness may be present. Inflation shows dilatation of the cecum and insufficiency of the ileocecal valves. X-ray discloses a dilated cecum in which bismuth may be retained for many hours.

Idiopathic Dilatation of the Colon.—Extensive and persistent dilatation of the colon results from a valve-like union of the sigmoid and rectum, or from a congenitally enlarged and misplaced colon. The characteristic finding is a large abdominal swelling due to gas, occurring practically always in children. The enlargement may be uniform or more marked on the left side of the abdomen. Passage of a rectal tube will relieve the distention. Subsequent inflation will outline the colon and cause a return of the swelling.

Simple Colitis occurs occasionally as an independent disease. It is manifested by acute diarrhea, blood-tinged stools with considerable mucus, local pain, and tenderness.

Physical Examination.—The patient presents the general features of the habitus. The position of the stomach can be accurately outlined only by gastric inflation. The greater curvature may be just below the umbilicus or it may extend down to the pelvis. A dilatation of the stomach walls is frequently found in association with the ptosis. The gastric contents show a diminution of free hydrochloric acid in cases without much dilatation. If there is much motor stasis, an actual achylia with retention will be found.

The diagnosis of gastroptosis is verified by the x-ray which shows the exact position of the stomach with remarkable clearness. Transillumination of the stomach may also be employed in order to get a clear outline of the position of the organ.

Nephroptosis.—This is a very frequent and commonly demonstrated condition. It must not be thought, however, because a kidney can be palpated that it will give symptoms. Only one case in ten gives the characteristic symptoms of dragging pain in the side, irritating urinary symptoms, gastric distress, and nervousness. If the kidney is freely movable there may occur intermittently a torsion of vessels and nerves with a resulting hydronephrosis. The resulting syndrome of intense pain referred toward the genitalia, nausea, vomiting, and signs of collapse is known as Dietl's crisis. With the passage of large quantities of urine, relief of the symptoms follow.

Examination.—The kidney should be first palpated with the patient lying down. To accomplish this the left hand is placed under the kidney pouch and is pressed upward. The right hand is then placed just below the costal margin and the patient is instructed to take a deep breath. At the height of inspirations the fingers are pushed deeply down under the costal margin. As the air is exhaled and the pressure slightly relaxed, the smooth surface of the kidney can be felt slipping under the fingers. Pressure upon the tumor is painful and causes a sickening sensation. If the kidney is extremely movable it may be necessary to employ the following procedure. The side is grasped by one hand, the fingers being beneath the kidney pouch and the thumb at the costal margin. Firm pressure is made by the thumb and fingers to prevent the passing of the kidney back into its bed. The abdomen is then either carefully palpated with the opposite hand until the kidney is found, or pressure is exerted upward by the palm of the hand, which will cause the kidney to slip under the thumb of the other hand. Palpation should be done with the patient in the erect posture or lying on the side. The kidney cannot be felt when the patient is in the prone posture. It is well before making the examination to have the patient move briskly around in order to displace the kidney somewhat.

Urine.—The urine shows nothing characteristic of the condition. The relief from an attack of Dietl's crisis is accompanied by an excessive flow of urine which may contain albumin and blood.

CHAPTER XLI

DISEASES OF THE LIVER AND PANCREAS

THE LIVER

THE symptoms of disease of the liver are due to (I) the morbid processes resulting in changes in anatomical structure of the liver; (II) disturbance of the functions of the hepatic cells; (III) obstruction of the channels for the flow of blood or bile.

I. Symptoms Due to Anatomical Changes.—The morbid processes cause anatomical changes in the size, shape, and consistence of the liver. For example, congestion of the liver results in an increased size of the organ; cancer, in alterations in the shape, and fatty infiltration in softening of the tissue of the enlarged palpable liver.

II. Symptoms Due to Functional Disturbances of the Liver.—The functions of the liver are (1) to secrete bile; (2) to render innocuous certain poisons; (3) to assist in the metabolism of proteins; (4) to store glycogen and to secrete it as glucose when required by the organism; (5) to store fat and to release it when the external supply becomes deficient; (6) to destroy hemoglobin and erythrocytes. Functional disturbances of the liver manifest themselves clinically by such symptoms as headache, lassitude, and often mental hebetude or pronounced excitement, due to incomplete destruction of poisons or by symptoms due to faulty metabolism, as wasting and cachexia.

III. Symptoms Due to Obstruction of the Channels.—Obstruction of the bile ducts or capillaries causes jaundice. Obstruction of the blood channels causes congestion of the liver.

Historical Diagnosis.—Primary disease of the liver is rare, secondary is common, therefore knowledge of possible etiological factors is of particular aid in the diagnosis of hepatic affections.

Social History.—Diseases of the liver usually occur late in life; sex is insignificant from a diagnostic standpoint, except that repeated pregnancy seems to have some influence in the formation of gall-stones. The habits of the patient are important; alcoholism points to cirrhosis, excessive use of stimulating foods to hyperemia.

Previous Disease.—The occurrence of heart disease or obstructive lung disease points to congestion; infectious disease to an otherwise unaccounted-for cirrhosis; dysentery to abscess; syphilis to cirrhosis, gumma, or amyloid disease; chronic suppuration to amyloid disease and pyemia; suppuration in the portal area to multiple abscess.

Subjective Diagnosis.—Pain, in congestion and carcinoma of the liver, is dull and aching, with a sense of distention in the right hypo-

chondrium. In acute perihepatitis it is sharp, lancinating, and exaggerated by breathing. In diseases of the bile passages it is sharp, colicky, and spasmodic, particularly in cholelithiasis. In chronic diseases of the liver, pain is usually absent.

Objective Diagnosis.—Jaundice.—Jaundice presents two forms: an *obstructive form*, the result of obstruction of the bile ducts, and the so-called *non-obstructive* or better *hemolytic form*.

GENERAL SYMPTOMS.—(1) Discoloration of the skin and mucous membranes; (2) discoloration of the secretions except the saliva, tears, and mucus; (3) absence of bile from the stools (clay-colored); (4) cutaneous irritaton: pruritus; (5) bradycardia; (6) tendency to hemorrhage; (7) cerebral symptoms of depression or extreme irritability.

OBSTRUCTIVE JAUNDICE.—1. *Jaundice from Disease Outside the Ducts.*—This may be due to extreme pressure upon the ducts by new growths in the liver or in the neighboring organs, particularly in the head of the pancreas, by accumulated feces in the colon, inflammatory adhesions, aneurism of the aorta, etc.

Jaundice of this form is recognized by its gradual onset, becoming progressively more marked; by the absence of pain; by the signs and symptoms of disease elsewhere, and by the absence of the usual signs of disease of the ducts. Occasionally kinking of the ducts as a result of ptosis of the stomach or liver cause jaundice.

2. *Jaundice from Disease of the Ducts.*—Catarrhal or suppurative cholangitis, cicatricial formations and cancer or other tumors involving the duct walls cause a stenosis of the biliary channels with a resulting jaundice. The diagnosis of this form of jaundice must be based upon the history and the symptoms of the causative factor.

3. *Jaundice from Obstruction within the Ducts.*—Gall-stones commonly and rarely worms, or other foreign bodies, are the cause of occlusion of the ducts. The intermittency of the jaundice, the occurrence of paroxysmal pain and intermittent fever are the diagnostic features of importance in this form of jaundice.

NON-OBSTRUCTIVE OR HEMOLYTIC JAUNDICE.—Various names have been applied to this form of jaundice, *e. g.*, toxic, infectious, hemato-hepatogenous, or toxemic obstructive jaundice. The important factor in the production of jaundice of this type is increased hemolysis, due to the action of hemotoxins. The mechanism briefly is as follows: Toxins of certain diseases or actual poisons introduced from without destroy the erythrocytes. As a result of the hemolysis an excessive amount of hemoglobin is liberated which causes an increased formation of bile pigments *by the liver*. This in time leads to pronounced viscidification of the bile, which from its thickness in turn causes a temporary obstruction in the bile capillaries, with a consequent absorption of the bile into the blood, the manifestation of this clinically by the presence of jaundice. increased viscosity of the bile may alone cause the jaundice, or to this may be added a partial obstruction by a descending catarrh of the bile ducts set up by the irritating action of toxic substances in the

itself. Hemolytic jaundice is not to be confounded with the obsolete term *hematogenous jaundice* in which the jaundice is supposed to be due to the pigments of blood destruction not altered by the liver. Bile pigment is always formed by the action of the hepatic cells, though Whipple has attempted to show that bile may be formed in the blood without the correlation of the liver. Jaundice of this form may be found in acute yellow atrophy, Weil's disease, pyemia, during the course of acute infectious diseases, as yellow fever, enteric fever, pneumonia, malaria and similar infections, or it may be the result of blood destruction by the action of certain hemolytic poisons, as phosphorus, chloroform, arsenic, or toluylendiamin. *Hereditary jaundice* is thought to be either the result of paroxysms of erthrocytolysis, as is the jaundice occurring in newborn babes (*icterus neonatorum*) or the result of congenital increase in the fragility of the red cells with persistent destruction of more cells than normally.

FEVER.—Rises in temperature of a varying degree are found in most of the affections of the gall-bladder and bile ducts. A normal or subnormal temperature is usually found in affections of the liver, except in the acute inflammatory processes, or occasionally in cirrhosis and the late stages of cancer.

Physical Diagnosis.—Changes in the shape, size, and position of the liver are recognized by physical examination and are discussed in the appropriate chapter. Inflammatory processes in the gall-bladder and the gall ducts are most often manifested by slight enlargement of the liver, by the increased size of the gall-bladder, by tenderness over the organ itself or at a point midway between the umbilicus and the ninth costal cartilage, and by the rigidity of the muscles of the right hypochondrium.

Laboratory Diagnosis.—*The Blood.*—An anemia is present when there is jaundice and is a sequel of the chronic hepatic diseases. In acute yellow atrophy there is often polycythemia with hemoglobinemia. A leukocytosis is present in the acute diseases of the liver and bile passages, except acute typhoidal cholecystitis and cholangitis which give a leukopenia. *The coagulation time* of the blood is increased from two to six minutes in jaundice.

Urine.—*Jaundice* results in a highly colored urine. Very moderate jaundice may at times be recognized only by the presence of bile pigments in the urine. Urobilinuria is commonly found in most diseases of the liver.

Functional Test.—Alimentary levulosuria is found as a result of disturbances of the hepatic function, notably in cirrhosis of the liver. (See Chapter XXX, page 450, for method.)

Deformities of the Liver.—**Linguiform Lobulation** (*Riedel's Lobe*).—This is an abnormal elongated lobe of the liver. A *corset liver* is the result of pressure by tight corsets, causing marked variations in the shape of the organ. These abnormalities are usually symptomless, although there may be attacks of pain and tenderness over the tumor,

is no visible evidence of superficial engorgement; (7) jaundice, the result of a catarrh of the bile ducts from interference with their circulation.

Thrombosis of the Portal Vein.—This may be due to the usual factors causing thrombosis, but most frequently is associated with cirrhosis of the liver or due to the pressure of intra-abdominal new growths upon the vein. It is a rare condition.

Symptoms.—The symptoms due to complete obstruction develop suddenly. There is often a sharp epigastric pain followed by hematemesis or melena. Ascites develops rapidly, is of extreme degree, and the fluid quickly reaccumulates after removal. The spleen is engorged and often enormously enlarged. The general condition rapidly becomes poor and cachexia soon appears.

Acute Hepatitis.—Acute hepatitis may be divided into the non-suppurative and suppurative forms. In the non-suppurative form the symptoms are virtually those of an aggravated congestion, but associated with fever. The suppurative form, or abscess of the liver, may be manifested either by a single abscess, the result of (1) amebic infection; (2) trauma from external sources; (3) parasites; (4) extension of suppurative process from neighboring organs; or by multiple abscesses, occurring: (1) secondarily to a general arterial pyemia; (2) secondarily to a portal pyemia, the result of inflammation of the portal vein, a suppurative pylephlebitis, arising from an ischiorectal abscess, appendicitis, or ulceration anywhere in the portal tract; (3) from extension of a suppurative cholangitis.

Single Abscess.—The general symptoms are those of suppuration; there is an intermittent fever, the rises in temperature being attended by chills and profuse sweats. There is pronounced malaise and prostration; jaundice may or may not occur while a leukocytosis is common. Locally there is pain over the liver referred to the right shoulder, which may be paroxysmal, or intense and persistent. A sense of weight and fulness in the right hypochondrium is noted by the patient who suffers also at times from dyspnea and cough.

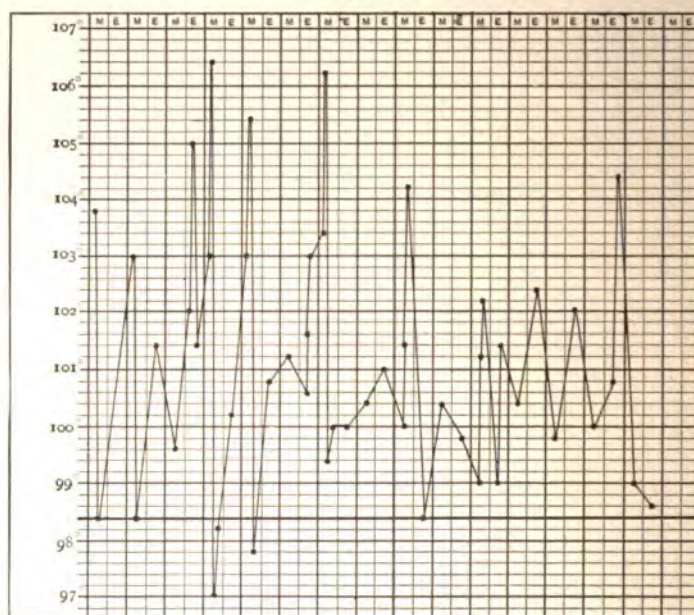
In *tropical (amebic) abscess* the commonest form of a single abscess, the general symptoms of suppuration and local symptoms of hepatic involvement may be absent for a comparative long period or there may be slight pain over the liver as the only local manifestation of the abscess, the general prostration and anemia of the dysentery obscuring the general signs of abscess formation.

PHYSICAL SIGNS.—The hepatic facies is characteristic. The skin is shallow, the complexion muddy, and the face pale. The facial aspect is one of anxiety and distress.

Examination of the liver by percussion demonstrates a uniform enlargement when the abscess is central. An abscess of the periphery of the right lobe (the usual site), according to its location, will cause irregular dullness, usually upward or more rarely, downward. This is often distinctly rounded in shape and associated with a corresponding

change in the area of hepatic dulness. Palpation over the irregular area of dulness causes pain. If the abscess is well advanced and adhesions have formed, the overlying skin may become reddened and edematous while fluctuation may be elicited; at times an audible friction sound may be heard as a result of the coincident perihepatitis. Abscess limited to the left lobe of the liver presents the same finding, except that the enlargement is to the left, and more frequently extends well into the epigastrium, causing marked prominence in the upper abdomen.

FIG. 194



Intermittent fever in abscess of the liver.

Perforation of Hepatic Abscess.—The first intimation of the existence of an abscess is sometimes obtained from the occurrence of rupture. The abscess most frequently perforates through the diaphragm and pleura into the lungs. An empyema is set up, with its characteristic physical signs. A cough develops and sooner or later typical sputum, likened to anchovy sauce, is expectorated. It is purulent in character and blood is mixed with the pus. Necrotic liver tissue may be found in the sputum or amebas, should they be the exciting cause. Rupture into the pericardium is usually followed by immediate death. Rupture into the peritoneal cavity causes acute peritonitis. Rupture into the stomach or intestines is recognized by the vomiting or pain in the stools of the brick-red "anchovy sauce" pus. With the occurrence of perforation there is a coincident subsidence in the size of the tumor and the appearance of the signs of shock.

DIAGNOSIS.—The diagnosis of simple abscess is based upon the discovery of a causative factor and the progressive enlargement of the liver associated with sepsis; of amebic abscess, upon the finding of amebas in the stools. The *x*-ray may aid in the diagnosis while exploratory puncture is a valuable procedure in many cases. The site of election is over the most prominent point of swelling, or if the swelling is not pronounced, in the lowest interspace of the anterior axillary line. A large caliber needle should be used. Pus, though present, is not always found, but if it should be discovered the diagnosis can at once be established. There are several conditions which may simulate a solitary hepatic abscess. One of these, malaria, is differentiated by the blood findings and by the disproportionate size in the liver and the enlarged spleen. So also *abscess of the abdominal wall* is differentiated largely by the absence of liver enlargement. A *subdiaphragmatic abscess* may frequently only be differentiated by surgical procedure, although at times the presence or absence of respiratory movement of the liver may be demonstrated; in a hepatic abscess the liver moves with respiration, while in a subdiaphragmatic abscess the liver is immobile.

Cholelithiasis is excluded by the absence of history of gall-bladder infection, by the absence of periods of apyrexia and by the presence of serious derangement of the general health.

Multiple Abscess of the Liver.—The symptoms of suppurative pyelphlebitis or of multiple septic pyemic abscesses of the liver are preceded by the symptoms of the disease to which it owes its origin. The liver is found uniformly enlarged and tender. Jaundice is more frequent than in solitary abscess, and to these local symptoms are added the constitutional symptoms of severe sepsis.

Acute Yellow Atrophy of the Liver.—An acute diffuse lesion of the liver with degeneration and necrosis of the cells, characterized by jaundice and cholemia, is most common prior to the thirtieth year. It is probably the result of autolysis, primarily instigated by a toxin having a specific action on the liver cells whose life it destroys without injury to its proteolytic ferments which in turn digest the dead hepatic cells with consequent development of a severe endogenous intoxication set up by the toxic products of the autolysis.

Many of the cases occur during pregnancy, while syphilis has also been found to have an important etiological bearing on the condition. The symptoms are local and general. It frequently begins with an attack of gastro-intestinal catarrh; it is often looked upon as a simple catarrhal jaundice, but in a week or ten days the patient becomes rapidly worse; headache and vomiting becomes prominent symptoms and hemorrhages are seen under the skin. Cerebral symptoms of delirium or stupor soon come into evidence and are rapidly followed by coma or by a profound typhoid state, with early exitus. The *urine* is scanty, bile-stained, and of high specific gravity. Leucin and tyrosin are always present, as well as diacetic and other fatty acids. The *stools* as a rule are dark colored and offensive, but may be clay colored, particularly

if there is diarrhea. *Examination* shows at first a slightly enlarged liver, which, however, so rapidly diminishes in size that in a short time the anterior dullness may be entirely obliterated. A concomitant enlargement of the spleen usually exists.

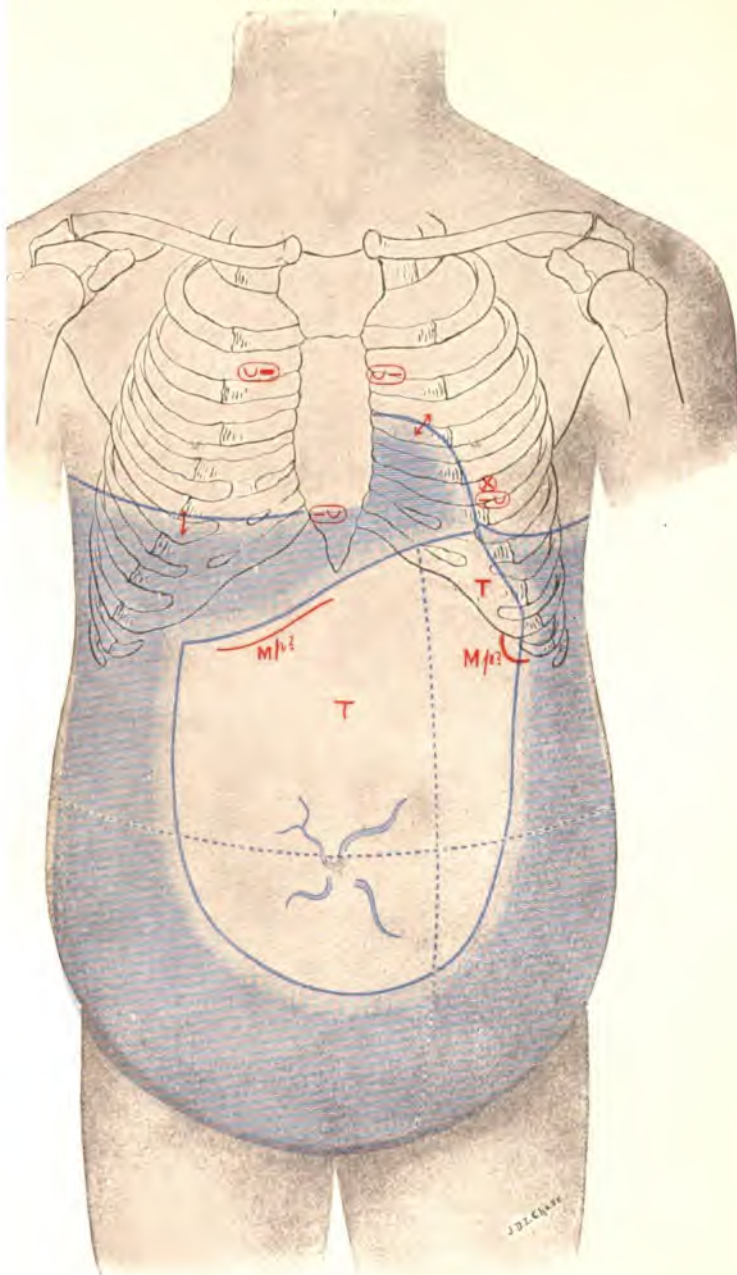
Diagnosis.—In the early cases the diagnosis is often made only by the finding of the characteristic changes in the urine. Later in the disease, the diminution in the size of the liver, associated with pronounced cerebral symptoms and the presence of leucin and tyrosin in the urine point unquestionably to the diagnosis. *Phosphorus-poisoning* is differentiated by the more pronounced gastric symptoms, particularly the severe vomiting, the vomitus often containing phosphorus; by the more persistent enlargement of the liver and by the absence of leucin and tyrosin. *Biliary cirrhosis* is excluded by the presence of an enlarged liver and spleen; by the more protracted course of the disease and by the absence of the typical urinary findings.

Chronic Interstitial Hepatitis.—Chronic interstitial hepatitis, or cirrhosis, is a diffuse interstitial inflammation of the liver. Two forms are recognized clinically: (1) portal (Laennec's, alcoholic or atrophic) cirrhosis; (2) biliary (Hanot's or hypertrophic) cirrhosis. In both forms the important etiological factor is the presence of some toxic or irritating substance within the portal circulation, although chronic biliary obstruction may sometimes play a part in the causation of the first type.

Portal Cirrhosis.—Alcohol is the most frequent and predominant cause of portal cirrhosis of the liver, while spices and condiments used to excess may also be causative factors. Infections of various kinds may be followed by cirrhosis. A malarial cirrhosis is frequently noted. Syphilis seems to render the liver particularly susceptible to cirrhosis, which may also be a sequel of scarlatina and other acute infectious diseases. Mechanical irritation by long exposure to certain dust particles, such as those causing pneumokoniosis, may produce an overgrowth of interstitial connective tissue. Metallic poisons, lead, silver, etc., as well as gout, and other metabolic diseases are of some importance in causing the condition. A secondary cirrhosis of the liver frequently arises from prolonged passive congestion or chronic obstruction of the bile ducts.

SYMPTOMS.—The disease may be symptomless throughout life, and may only be discovered after death from some other cause. Again, no symptoms may be present until the sudden occurrence of hemorrhage from the stomach or bowel suggests the possibility of cirrhosis. When symptoms are present they may be roughly divided into those occurring while the collateral circulation is efficient, the first stage, and those occurring after failure of collateral circulation, the second stage. The first stage symptoms are simply the expression of a gastro-intestinal catarrh, as a coated tongue, irregular appetite, morning eructations, constipation, and loss of weight. As the disease progresses the above symptoms become more marked, the collateral circulation fails, and portal congestion develops with its train of symp-

PLATE XXV



Cirrhosis of the Liver with Ascites.

toms. (See page 700.) Toxic symptoms may develop, at times, in the late stages. They are manifested by mental hebetude, delirium, or even convulsions.

The general symptoms of cirrhosis, and particularly those of the later stage, are striking and diagnostic. The patient, who in a large majority of cases has been corpulent, becomes emaciated; the skin changes in color and becomes an earth-gray or a dirty, sallow hue; the capillary venules of the faces are dilated. Later, ecchymoses may develop under the skin, and hemorrhages take place from the mucous membranes. Debility ensues; ascites develops; edema of the ankles appears and sometimes general anasarca may take place. Fever is rare. The *urine*, as ascites develops, becomes scanty, dark in color, and contains an abundance of urates and uric acid. In the so-called *bronzed diabetes* (cirrhosis of hemochromatosis) sugar is present, not as a result of the fibrosis of the liver, but as a result of the concomitant fibrotic changes in the pancreas. The blood findings are those of a secondary anemia.

PHYSICAL SIGNS.—The liver is enlarged in most cases, somewhat tender, and has usually an irregular surface. Even after ascites has become pronounced, the liver is often still found enlarged. In the late stages the area of hepatic dulness may be much diminished. The spleen is slightly enlarged and palpable. Ascites may be so severe that only after paracentesis can the size of the liver be determined.

DIAGNOSIS.—In the early stages the diagnosis must be based largely upon the history, so often of continued use of alcohol. Functional tests of hepatic efficiency are valuable adjuvants to other early symptoms and findings.

Chronic peritonitis with large amounts of fluid must be differentiated from portal cirrhosis with ascites. If there is any question of the diagnosis, the exudative fluid of peritonitis can be readily distinguished from the transudative fluid of cirrhosis by laboratory examination. However, the comparative frequency of tuberculosis of the peritoneum occurring as a complication of cirrhosis must be borne in mind in studying the fluid, and due allowances must be made for this possibility, which can only be accurately demonstrated by animal inoculation. Other conditions associated with abnormal fluid in the abdomen, *e. g.*, tumors of the *peritoneum*, *perihepatitis*, *thrombosis of the portal vein*, and *chronic cardiac diseases*, usually present no difficulty in differentiation from cirrhosis if a careful study of the case is made. An enlarged cirrhotic liver without ascites must be differentiated from other enlargements of the liver, which, however, are always secondary, so that the discovery of the primary cause of the enlargement will immediately clear up the diagnosis. Mild hematemesis and other gastro-intestinal symptoms of portal cirrhosis may suggest a *gastric ulcer* or *carcinoma*, which possibility can be excluded by analysis of the gastric contents.

Biliary Cirrhosis.—Chronic interstitial hepatitis of this rare form is in all probability the result of a pericholangitis of infectious origin and in but few ways resembles the common form of cirrhosis. The disease is

of the organ are somewhat soft at first, although later they may become hard and indurated.

Amyloid Degeneration of the Liver.—This disorder, usually the result of prolonged suppuration, may occasionally follow cachectic states. The symptoms are those of amyloid disease in general, progressive emaciation, anemia, and debility. The liver is much enlarged, smooth and indurated. The spleen is enlarged and the urine albuminous and abundant.

Carcinoma of the Liver.—Cancer of the liver in the vast majority of cases is secondary to cancer in some other location, as the rectum, colon, uterus, stomach, or the head of the pancreas. In many instances secondary cancer of the liver may be present without symptoms referable to that organ and is discovered only at autopsy. The symptoms are insidious in onset. Emaciation and loss of strength may be present for some time before the real cause of the cachexia is recognized. Fever of a moderate degree is frequently found, particularly in case of rapidly progressing tumor. Local discomfort is quite common but actual pain is usually absent. Jaundice is present in more than half the cases and ascites only slightly less frequent. The two in combination occur in about one-fifth of all cases.

Physical Signs.—Upon palpation the abdomen is distended and the superficial veins prominent; the liver is found enlarged and its surface irregular. The edges are uneven and nodules can be distinctly felt on the surface. They are usually hard, but as the disease progresses may become soft and even fluctuate. Enlargements of the left supraclavicular glands can often be palpated and are a manifestation of metastasis. Progressive enlargement of the liver is a diagnostic finding of importance. The enlargement can be well-defined by percussion; and, while the surface is irregular, the general shape of the dullness corresponds to that of the liver. In rare instances, as when the cancer is associated with advanced cirrhosis of the liver, the enlargement may not be found.

Diagnosis.—In rare cases in which the liver is smooth, cancer may be mistaken for a fatty or amyloid liver. Careful search, however, will usually assign a definite cause for the latter conditions.

A *syphilitic liver* with irregular gummata may be readily differentiated by the positive specific serological test.

In *biliary cirrhosis* the jaundice is deeper and persistent with but slight wasting or anemia. The surface of the liver is always smooth and without the characteristic nodular irregularities of cancer. In *portal cirrhosis* one finds more pronounced symptoms of portal obstruction, a history of alcoholism, and an enlarged spleen.

Suppuration of one or two carcinomatous nodules may simulate *abscess of the liver*, but cachexia is more marked and jaundice or ascites more frequent in cancer than in abscess. Cancer of the liver may be simulated by cancer of organs in close proximity to the liver, as the pyloric end of the stomach, the pancreas, and the colon. In connection with the first of these one finds in addition to the usual symptoms of pyloric cancer that jaundice occurs late. Also cancer of the pyloric

Physical Examination.—If the cysts are superficial when palpated with the fingers of the left hand and percussed with the right hand, a trembling movement which may continue for a certain time is felt (hydatid fremitus). Fluctuation may also be elicited. Cysts deep in the liver tissue give an elastic feeling to the palpating hand.

Diagnosis.—The occurrence of irregular painless enlargements of the liver without general symptoms points strongly to hydatid disease. If the hydatid fremitus is detected, a more positive conclusion can be reached. An absolute diagnosis can be made if by exploratory puncture the hooklets are found in the aspirated fluid. If sugar is found in the fluid a strongly presumptive diagnosis may be made. At times the disease may have to be differentiated from a syphilitic hepatitis after healing of gummata, from an enlarged gall-bladder containing fluid, or from hydronephrosis.

DISEASES OF THE GALL-BLADDER AND GALL DUCTS

Acute Catarrhal Cholangitis (Catarrhal Jaundice).—**Causes.**—Secondary to gastroduodenal catarrh, to acute infectious diseases, to pressure, to local spreading infection, and rarely to a primary infection, epidemic in type.

Symptoms.—The symptoms and signs of jaundice are well-known. These with the age of the patient, the presence of a cause, and the clinical course make up the picture. When long-continued, catarrhal inflammation may resemble obstruction due to other primary or secondary processes.

Diagnosis.—The diagnosis of catarrhal jaundice in cases continuing more than six weeks should be revised if the number of erythrocytes suddenly diminishes, the hemoglobin falls, the spleen enlarges, and there is loss of weight. The true nature can be determined by the antecedents or by accompaniments, which by this time may be more prominent, as the enlarged glands of syphilis or tuberculosis. Organic diseases, cirrhosis, cancer of the liver, and infections, as Weil's disease, must be excluded. Many times the catarrhs are the result of typhoid, pneumococcus, or other infections.

Suppurative Cholangitis.—The disease is a result of virulent bacterial infection of the biliary ducts and as a rule follows disorders that interfere with the circulation of the bile. The diagnosis is based on the:

1. **Clinical Course.**—Gradual onset; infection of ducts or gall-bladder; presence of foreign bodies (gall-stones); history of a previous general infection.

2. **Objective Diagnosis.**—Icterus may or may not be present. The fever is characteristic (hectic type). The toxemia due to the jaundice gives rise to toxic symptoms.

3. **Physical Diagnosis.**—The liver is moderately enlarged and tender. There is also a tender area in the region of the twelfth dorsal vertebra, 2 to 3 cm. from the median line (Boas).

4. **Laboratory Diagnosis.**—Leukocytosis is present. The Widal reaction should be taken to exclude typhoid fever and the blood examined to exclude malaria.

Differential Diagnosis.—From pylephlebitis the distinction is made by the absence of any cause for infection in the portal area; amebic abscess is excluded by the clinical antecedents, the physical signs, and the absence of leukocytosis; from malaria by examination of the blood; and from simple cholelithiasis by the presence of leukocytosis.

Cholecystitis.—Acute cholecystitis is usually due to an infection of the gall-bladder, the resistance of which is lowered by the presence of gall-stones, although it may be the result of bacterial invasion of a normal gall-bladder in the course of acute infectious disease, notably typhoid fever, or it may follow an acute gastroduodenitis. There are recognized several types, the catarrhal, suppurative, and phlegmonous.

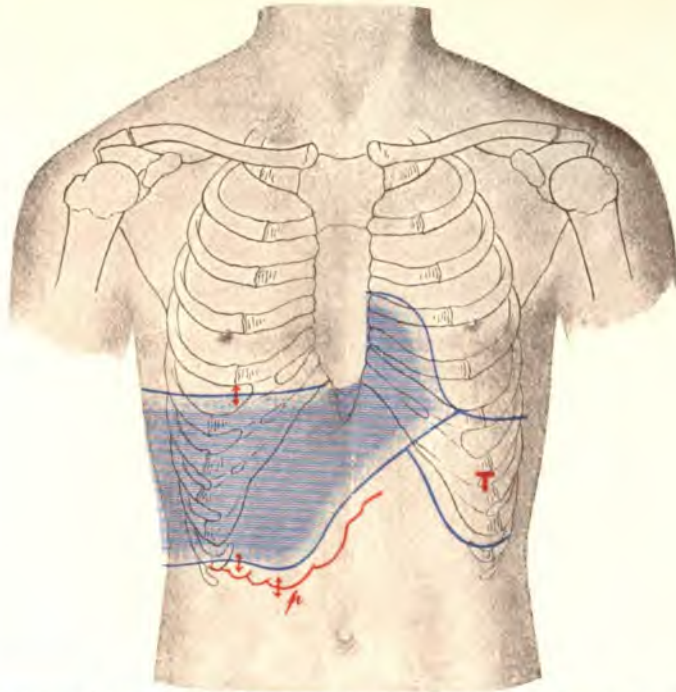
Symptoms.—The symptoms of acute catarrhal cholecystitis are frequently marked by the symptoms of the overlying condition. When symptoms are manifest, they are usually ushered in by nausea and vomiting, with pain occurring at the same time or shortly afterward. The pain is sharp, moderately severe, and subject to acute exacerbations. At first it may be in the epigastrium, but it soon becomes localized in the right hypochondrium. The pain may be referred toward the right shoulder or downward toward the right lower abdominal quadrant. Jaundice is absent unless there is an accompanying cholangitis. Fever is usually mild and general symptoms are not pronounced unless the infection of the gall-bladder occurs during convalescence from typhoid or other acute infectious disease, when the local and constitutional symptoms are frequently severe. Examination will show an enlarged gall-bladder over which there is distinct tenderness and rigidity of the muscles. Blood examination shows a polynuclear leukocytosis.

Acute suppurative and *acute phlegmonous* inflammation of the gall-bladder present more marked local symptoms and more pronounced general symptoms with greater prostration than is found in the acute catarrhal inflammation. Peritonitis ushered in by severe pain and shock rapidly ensues unless surgical relief is given.

Cholecystitis in Typhoid Fever.—This develops in the third or fourth week of the disease or in the period of convalescence. The onset is characterized by an increase in the temperature with synchronous severe pain in the region of the gall-bladder and localized tenderness and rigidity of the right rectus muscle. The severity of the attack varies. It may be so mild that the local symptoms are not recognized and the patient is thought to have a relapse. Occasionally it may be fulminating in character and be rapidly followed by perforation of the gall-bladder. A primary typhoid cholecystitis frequently associated with cholangitis may occur without any direct evidence of a general typhoid infection. In most cases of typhoid fever the gall-bladder contains the typhoid bacillus, but only in rare cases do symptoms acute cholecystitis develop. The typhoid infection may be manifest

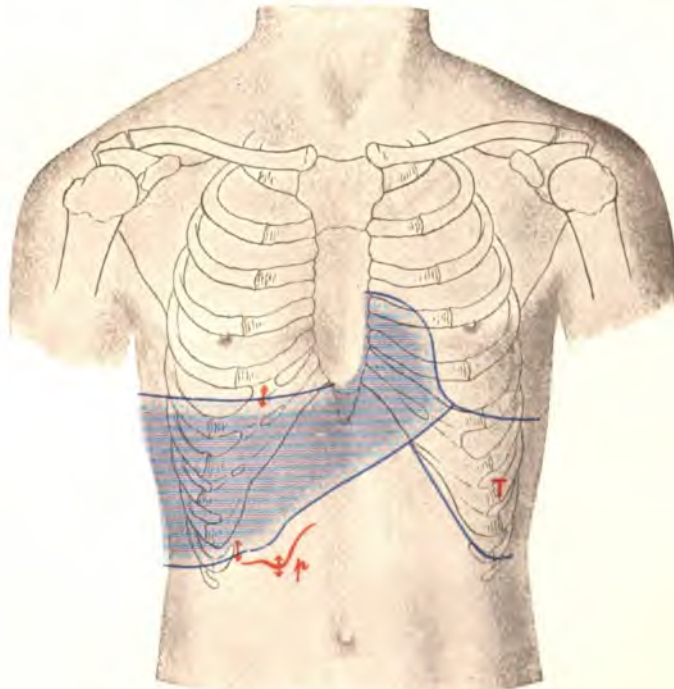
PLATE XXVI

FIG. 1



inoma of the Gall Bladder with Involvement of the L

FIG. 2



Enlargement of the Gall Bladder

long after the subsidence of the original infection by the development of symptoms of gall-stone which have formed as a result of the earlier involvement of the gall-bladder. In many cases the typhoid bacilli persist in the gall-bladder for years without symptoms, as the so-called carrier had developed an immunity which prevents his reinfection. Such carriers, however, periodically or continually discharging the typhoid bacilli in the stools, are a constant menace to the health of the community.

Differential Diagnosis.—Atypical forms of *acute appendicitis* or *cholecystitis* may so simulate each other that a differentiation can only be made by a careful study of the symptom-complex, and a thorough review of the past medical history. In a similar manner *acute intestinal obstruction* or *perforation of a gastric ulcer*, may simulate or be simulated by the more severe forms of cholecystitis. *Multiple abscesses* of the liver or *pylephlebitis* are differentiated by the antecedent history of abdominal infection, by the enlargement of the liver, and the signs of portal obstruction.

Amebic abscess of the liver is usually differentiated from cholecystitis by its slower course, by the enlargement of the spleen, and by the discovery of the amebas in the stools.

Chronic Cholecystitis in the vast majority of cases is associated with gall-stones. The symptoms are so similar to those of cholelithiasis that it is impossible to tell whether or not gall-stones are present.

Primary Cancer of the Gall-bladder and Biliary Ducts is comparatively rare, but four times more common in the former location than the latter. Cancer of the gall-bladder is commonly associated with cholelithiasis and the early symptoms are usually those of gall-stones. Later continuous pain with acute exacerbations is a prominent symptom, and with it appears jaundice. The gall-bladder becomes enlarged, palpable, and moderately tender; still later it may become irregular and nodular. The general health fails and cachexia now develops rapidly. Primary cancer of the biliary ducts is rare. Persistent jaundice in a person over forty should create suspicion of the true nature of the condition. With the appearance of profound anemia and cachexia the diagnosis is assured.

Cholelithiasis.—Naunyn's theory of the formation of gall-stones is the one generally accepted. Through the agency of various organisms a catarrh of the mucous membranes is set up. This leads to a stasis of the bile with increased precipitation of cholesterin and calcium salts, exfoliation of the epithelial cell and consequent formation of gall-stones. Another recent theory, a revival of an old assumption, is that the formation of gall-stones depends upon biliary stasis associated with disturbance of cholesterin metabolism. The believers in the first theory point to the frequent occurrences of gall-stones after gall-bladder infections as one of the many arguments in their favor, while those that believe in the latter theory point to the frequent tendency of gall-

pain is usually severe, paroxysmal, and located in the right hypochondrium or the epigastrium. It radiates around the chest or is referred to the right scapular region. The pain may be intermittent; it may cease suddenly or the agonizing quality may become a dull, steady ache to subside more slowly. In many instances a chill precedes or immediately follows the pain, after which the temperature rises for a short time and then disappears rapidly unless an acute inflammation of the gall-bladder is set up. Jaundice is a common symptom. A leukocytosis may occur during an attack and is probably the result of some acute inflammatory process in the gall-bladder. The bowels are not disturbed during an attack, and their color remains normal unless there is an obstructive jaundice. Frequently upon straining the feces through coarse gauze, gall-stones may be found. The urine is high colored, and may contain bile.

Examination discloses considerable tenderness in the right hypochondrium and more marked over the gall-bladder. The tenderness persists for some time, although the pain may disappear rapidly. At times the enlarged and tender gall-bladder may be palpated, and if the walls are not too tense a gall-stone crepitus may be elicited. A special train of symptoms arises if a gall-stone becomes lodged in the cystic or common duct. Gall-stones may also result in certain accidents with their peculiar symptoms.

Obstruction of the Common Ducts of Gall-stones.—A calculus may lodge in the common duct and cause complete obstruction with the production of permanent jaundice. More frequently, however, it lodges in the diverticulum of Vater and acts as a ball-valve so that an intermittent obstruction is produced. This latter form of obstruction is usually associated with a concomitant cholangitis, so that paroxysms of intermittent fever (the hepatic intermittent fever of Charcot), chills and sweats, occur, together with intermittent or remittent jaundice. Hepatic colic may occur at the same time or there may be simply mild attacks of pain associated with gastric disturbances. The pain is often relieved by vomiting or by certain postures of the body. Gall-stones lodged in the common duct may cause suppuration, with secondary extension of the inflammation to the smaller ducts and the gall-bladder.

Obstruction of the Cystic Duct.—A gall-stone lodged in the cystic duct may cause a simple acute or even a suppurative cholecystitis. As a rule, however, infection is absent or mild and soon subsides. The obstruction then, if complete, results in a chronic dilatation of the gall-bladder, hydrops or dropsy of the gall-bladder. The enlargement is noted at the edge of the liver in the usual situation and may gradually increase to an enormous extent, at times projecting downward as far as the groin. The tumor is movable with respiration, and the shape is globular, conical, or pear-shaped. It is usually tender and firm, but elastic on pressure. Fluctuation may be detected. Jaundice is absent as are other symptoms except those that result from the presence of the tumor. When the obstruction is not complete, a chronic atrophic

late life, and appears frequently without apparent cause. The patient is seized with severe pain localized in the upper abdomen, which may be intermittent like colic; nausea and vomiting take place almost at the same time. Collapse with subnormal temperature ensues shortly. The general condition gets rapidly worse, terminating in death in from twenty-four to seventy-two hours after the initial pain.

Acute Hemorrhagic Pancreatitis.—This occurs more frequently in men than women. Injury, cholelithiasis, and cholecystitis are the important etiological factors. The disease is a result of the introduction of some substance, most often bile, into the secretory parenchyma, causing a necrosis of the cells with a resulting hemorrhage. It is quite possible that the introduction of the irritant foreign substances sets up a necrosis of the pancreatic substance by the action of the pancreatic secretions themselves. The severe shock is thought to be a manifestation of toxemia produced by the toxic action of the products of autolysis of the pancreas.

Symptoms.—The attacks develop suddenly with violent abdominal pain localized around the umbilicus; nausea and vomiting are early present in all cases and likewise constipation in about one-half of the cases. The symptoms of shock develop suddenly and a subnormal temperature is usually found. Jaundice and glycosuria may be present in a small percentage of cases. The abdomen is distended, some tenderness may be elicited in the epigastrium and a vague tumor mass may be palpable in the region of the pancreas. Leukocytosis is usual. The cases that survive the fourth day of the disease show a moderate diarrhea, a lessening of the severe pain, elevation of temperature, and a distinct tumor in the lower epigastrium extending toward the spleen and down toward the umbilicus. In these cases a gangrenous pancreatitis has developed and the symptoms become the general ones of sepsis plus its local manifestations.

Diagnosis.—Acute hemorrhagic pancreatitis may resemble acute intestinal obstruction, intoxication with irritant poisons, hepatic colic or perforation of the gastro-intestinal or biliary tract. The difficulty of diagnosis, however, is so great that resort to laparotomy is justifiable in order to determine the exact nature of the condition.

Suppurative Pancreatitis.—Adult males under forty years are most frequently the subject of this condition. It usually occurs as a complication or sequel of some primary pancreatic or peripancreatic disease. The symptoms continue during several weeks and may even persist for as long as a year. Pain in the epigastrium, associated with irregular vomiting, is the usual picture. Fever is irregular and chills usually precede the elevation of temperature. Jaundice is frequently, and glycosuria and fatty diarrhea rarely, found. Progressive emaciation and exhaustion develop more or less rapidly. Examination will show a tumor occupying the position of the pancreas, often fluctuating and frequently painless.

Chronic Pancreatitis.—The symptomatology of chronic pancreatitis is vague and indefinite. The symptoms are those of an indigestion, characterized principally by irregular vomiting, constipation, and by discomfort some time after eating. Emaciation is usually pronounced. A somewhat more definite sign is an epigastric mass that can sometimes be distinguished from the stomach. Determination of impairment of the pancreatic function affords the most valuable aid in arriving at a diagnosis of chronic pancreatic disease. The functions of the gland are the secretion of ferments concerned in the digestion of carbohydrates, fats, and proteids, and the elaboration of an internal secretion which plays an important part in the metabolism of sugar. Disturbances of function then are recognized clinically by the occurrence of intestinal indigestion, by emaciation, by the presence of excessive fat and undigested meat particles in the stools and at times by the presence of glycosuria. The early diagnosis depends upon the evidence of pancreatic insufficiency as afforded by the various laboratory tests; late diagnosis is aided by the presence of fatty stools and glycosuria. (See Chapter XXX, page 450.)

Cysts of the Pancreas.—These are not always true cysts but may be an encapsulated accumulation of fluid in contact with the pancreas and apparently derived from it. A true pancreatic cyst may develop as a result of obstruction of the pancreatic duct, or from trauma, or as a true proliferative cyst.

Symptoms.—There is a sense of weight and fulness in the epigastrium, slight jaundice and frequently light colored stools. Pain is characteristically present. Alterations in the pancreatic functions are usually absent and depend upon the extent of destruction of the gland.

Physical Signs.—A tumor of the lower epigastric and upper umbilical region of the abdomen, extending somewhat to the left of the midline, is typically found. The tumor is smooth, elastic, soft, lobulated, and may fluctuate. It moves with respiration. The tumor may dislocate the heart upward, displace the stomach toward the right, push the colon downward, and the diaphragm and the contents of the chest upward.

Diagnosis.—Exploratory puncture may establish the diagnosis, but is usually so unsafe that it should be resorted to only in extreme cases. Pancreatic cysts must be differentiated from cysts of neighboring organs, from cancer of the pancreas, aneurism, hydronephrosis, and circumscribed peritonitis with exudation. The location of the cyst is best determined by its relation to the stomach and the colon. If the stomach and colon are outlined by auscultatory inflation, the cyst will be found to lie between the two organs; if inflated, percussion will show that the cystic dulness is replaced by tympany. Cysts of other organs present different relations to the colon and stomach than does a pancreatic cyst. Dulness over hydatid cyst of the left lobe of the liver is continuous with that of the liver, but the dulness of a pancreatic cyst and the liver is interrupted by gastric tympany.

Cancer of the pancreas is usually not differentiated from a small cyst until the occurrence of cachexia. *Aortic aneurism* is differentiated from a pulsating cyst by placing the patient in the knee-chest position, when the pulsation will cease.

A hydronephrosis of the left kidney is lower in the abdomen and does not have the same relation to the stomach and colon as a pancreatic cyst.

Pancreatic Calculi.—These are extremely rare. Their presence is very occasionally recognized when their passage causes severe colic. The pain is sudden, severe, and sharp. It is referred along the left costal margin to the left scapular region. Nausea and vomiting with signs of mild prostration are noted. Glycosuria and steatorrhea are usually present. A positive diagnosis is established if a small, white, hard, irregular, and jagged stone is found in the stools.

Carcinoma of the Pancreas.—Cancer of the pancreas occurs more commonly in the head than in other parts. The early symptoms are severe and deep-seated epigastric pain and intestinal indigestion. Later bulky, fatty stools and glycosuria are present. Cachexia develops rapidly. Progressively increasing jaundice, the result of pressure on the bile ducts, is a frequent and important symptom.

Physical Signs.—A tumor of the epigastrium may be made out if the patient is much emaciated. This tumor may simulate an aneurism, but is distinguished by the up-and-down movement of the hand placed over the tumor rather than the true expansile pulsation of an aneurism. A tumor of the pylorus is excluded by the more superficial location and the presence of pyloric obstruction. A tumor of the transverse colon is differentiated by its movability, its association with intermittent constipation, and the occurrence of intestinal hemorrhage.

CHAPTER XLII

DISEASES OF THE KIDNEYS

General Considerations.—Diseases of the kidney may be the result of:

1. Congenital abnormalities, which are relatively frequent in the kidneys and may cause renal disease.

2. Circulatory derangements, as hyperemia and congestions, thrombosis and embolism, which are common, owing to the very abundant blood-supply of the organ.

3. The action of infectious material (bacterial), ingested exogenous toxins and endogenous toxins, and waste products of protein metabolism, which are eliminated by the kidneys. On account of its function the organ is likely to be affected by the presence of infective and irritative substances in the economy, causing acute and chronic inflammatory lesions.

4. Ascending infections from, or mechanical or functional disturbances of, the lower urinary tract.

5. Malignant disease.

Anatomical and Physiological Symptoms.—The symptoms of renal disease are due to (1) the morbid processes; (2) anatomical alteration of the organ; (3) perversion of the renal function. The symptoms of the morbid processes are the general ones that arise from similar changes anywhere in the economy. The anatomical changes in the kidney, for obvious reasons, are of little value, as only pronounced enlargement of the organ is recognizable subjectively or objectively. It follows then that the important signs and symptoms of renal disease are those that arise as a result of alterations or perversions of the renal function of excretion. These symptoms are (1) alterations in the urine; (2) edema; (3) anemia and wasting; (4) uremia; (5) cardiovascular changes; (6) retinal manifestations.

Alterations in the Urine.—These include variations in the normal constituents of the urine and the presence of abnormal constituents in the urine. The gross clinical manifestation of variations in the normal constituents of the urine is an increase (polyuria) or decrease (oliguria) in the amount of urine passed in twenty-four hours, as estimated accurately by measuring the total amount passed in the period. At times polyuria is noted by the patient as a result of increased frequency of urination during the day or as a result of the necessity arising at night to void the urine. Increased frequency due to disease of the bladder, to diabetes, to reflex irritation by a ureteral calculus, to a neurosis, or to concentration of the urine must, however, be eliminated before it is attributed to renal disease. In Bright's disease

polyuria is an accompaniment of the chronic inflammatory conditions, more particularly chronic interstitial nephritis, and is due largely to increased blood-pressure, while oliguria is found in acute, subacute, and in chronic parenchymatous nephritis and in congestion of the kidneys, in which conditions the rate of the flow of the blood through the kidney is retarded and the renal blood pressure is lowered. Variations in the excretion of nitrogen compounds, chlorides, sulphates, and other constituents of the urine are also found in most diseases of the kidneys.

The abnormal constituents of the urine found in Bright's disease, including albumin, tube casts, blood, and pus, are discussed in Chapter XXVII.

Edema.—(See Chapter XIV.)

Anemia and Wasting.—Severe anemia is a frequent symptom of nephritis. It is associated with the peculiar pallor of kidney disease. The face is pallid and of a waxy whiteness, which, if the disease is of long standing, gives way to an ashen gray or sallow tint and the skin becomes dry and harsh; more or less pronounced wasting is also seen. The anemia and wasting are probably due in part to the increased elimination of protein, to the increased disintegration of the protein tissues (Bradford), and to the secondary gastro-intestinal complications.

Uremia.—Diseased kidneys do not eliminate the poisonous products of tissue-waste. The toxic matter is retained within the blood and produces a toxemia which may be acute or chronic. In acute uremia the manifestations develop suddenly and continue but a short period of time with alarmingly active symptoms, until death or recovery. In chronic uremia the onset is gradual and the manifestations may be limited to one or two conditions, as headache or morning nausea, which disappear and recur, or they may include the more pronounced symptoms of uremia. *Latent uremia* is seen in calculous suppression.

Nervous Symptoms.—*Headache.*—The pain is situated in the occipital region and may extend down the neck. It is severe and of a bursting character, and may be associated with giddiness. In both acute and chronic nephritis, headache is often the first manifestation; it may be associated with eye symptoms; it may be present on waking, and continue only through the morning hours. In acute uremia it persists throughout the attack.

Delirium—The delirium may be mild, but is sometimes attended by delusions, often with subsultus and picking at the bedclothes. Again, the delirium may amount to true mania, and the patient may exhibit other maniacal symptoms. On the other hand, the patient may be noisy, restless, and sleepless. Melancholia and delusional insanity may develop after the violent nervous symptoms of uremia have passed off.

Convulsions.—A convulsion may be the first indication of disease of the kidneys, or it may succeed a few days of persistent headache, or follow an attack of uremic vomiting. The convulsion resemble

epilepsy, and hence is known as an epileptiform convulsion. Sometimes a focal or Jacksonian epilepsy occurs instead of the true epileptiform convulsion. Temporary blindness may follow the convulsion (uremic amaurosis), and uremic deafness may occur.

Coma.—After the convulsion consciousness may return, or the patient may lapse into stupor, followed by complete coma. Coma may develop without convulsions, or immediately succeed a general convulsion. Headache or eye symptoms may precede the coma.

Local Palsies.—Dercum was among the first to call attention to the occurrence of uremic monoplegia or hemiplegia. Palsy develops suddenly and may occur after a convulsion. *Cramps* in the muscles of the calves, particularly at night, are of common occurrence.

Pruritus, local and general, is another nervous symptom that may be of uremic origin.

Pain in the upper abdomen, particularly in the median line, is a frequent precursor of more severe uremic symptoms.

Respiratory Symptoms.—*Uremic Dyspnea.*—Modifications of the breathing often accompany symptoms of uremia. The dyspnea may be constant or it may occur in paroxysms, or the two types may later alternate. A common type in uremia is the Cheyne-Stokes breathing. Paroxysmal dyspnea usually occurs at night, and resembles true bronchial asthma in every respect.

In addition to dyspnea, the occurrence of inflammatory pulmonary complications may be the first indication that the condition of the urine should be inquired into. Bronchitis, pneumonia, and pleurisy are common complications of uremia.

Gastro-intestinal Symptoms.—Loss of appetite is common. It is attended with absolute distaste for food after a small portion has been taken. Nausea may be continuous, or more frequently limited to the early morning. Vomiting may be paroxysmal, occurring chiefly in the early morning; or it may be sudden in onset, uncontrollable, and continue until nervous symptoms of uremia develop. Constipation is the rule in the course of chronic Bright's disease. One of the manifestations of uremia is diarrhea, often in the form of sudden, profuse, serous purging. Hiccough, although a muscular affection, is usually associated with gastric disturbances. Gastralgia, chronic gastritis, enteritis, and ulcerative colitis are of fairly common occurrence in chronic uremia.

Cardiovascular Symptoms.—The cardiovascular changes in nephritis are extremely important, and involve both the heart and the vessels, large and small. The cause of these changes has been variously explained, but no one theory has met with universal acceptance. The changes are probably the direct result of high blood-pressure, which in turn may be due to the fact that the filtration through the glomeruli is dependent upon blood-pressure, so that when the glomeruli are diseased greater pressure is required, *i. e.*, a compensatory rise in pressure develops.

The high blood-pressure has also been explained on the basis of an excessive amount of the secretion of the adrenal glands in the blood, the result either of increased secretion, the result of the nephritis, or increased retention of adrenalin from failure of elimination by the kidney.

The hyperpiesis may also be the result either of constriction of the capillaries or increased force of the heart, stimulated by the retained toxic products of nitrogenous metabolism.

Hypertrophy of the Heart.—The most pronounced change is hypertrophy of the left ventricle, which in long-standing cases is followed by hypertrophy of the other ventricle. The primary hypertrophy is later associated with eccentric hypertrophy, *i. e.*, hypertrophy with dilatation. Pronounced dilatation of the heart is usually a terminal finding in the course of chronic nephritis, and is the result of degenerative myocardial changes.

Arterial Changes.—The arterial changes are those of arteriosclerosis and atheroma. These changes are preceded by pronounced elevation of the blood-pressure, which is a usual and extremely frequent accompaniment of nephritis. Miliary aneurisms of the arterioles frequently develop, particularly in the brain, where the rupture of one of them may result in a terminal cerebral hemorrhage. In addition, rupture of an arteriole may cause epistaxis, retinal hemorrhage, less frequently hemorrhage from the bowels, subcutaneous hemorrhage, and hemorrhage from the pelvis of the kidney (renal epistaxis).

In addition to high arterial tension and accentuation of the aortic second sound, the objective symptoms of atheroma of the aorta and arteries are associated with chronic inflammation of the kidneys.

Here may be mentioned other relations of the heart and kidneys: (a) We have renal disease following forms of cardiac disease. In dilatation of the heart passive congestion of the organs takes place. The kidneys quickly become the seat of such congestion. In the course of simple dilatation or of valvular heart disease, the secondary dilatation, passive congestion, and chronic inflammation develop slowly. Embolism may also occur. (b) Renal disease, arteriosclerosis, and cardiac disease may develop at the same time from a common cause as alcoholism, gout, etc.

Retinal Changes.—The eye-grounds should always be examined; indeed, the patient himself by his complaints often directs attention only to the eye, the examination of which discloses the presence of an albuminuric retinitis. The following changes may occur in the acute or chronic forms of nephritis, although they are more common in the latter; (1) a diffuse, slight opacity and swelling of the retina, due to edema; (2) white spots or patches of various sizes, for the most part the result of degenerative processes; (3) hemorrhages; (4) inflammation of the intra-ocular end of the optic nerve; (5) atrophy of the retina and nerve may sometimes result from and succeed the inflammatory changes. These changes may affect one eye only (Gowers). It must

not be forgotten that temporary blindness may occur independently of retinitis.

Physical Diagnosis of the Kidney.—Renal Tumor.—Enlargements of the kidney may be detected by percussion. As the causes which produce enlargements of the kidney sufficiently great to be detected by percussion do not, with rare exceptions, involve both kidneys at the same time, comparison of the two sides is of great value.

Renal tumors give rise to swelling in the loin with muscle resistance if there is inflammation. They may extend forward, and are then palpable between the ribs and the transverse umbilical line. The mass is never notched, is usually smooth, and often takes the shape of the kidney if that organ is involved in its entirety. Otherwise the outline is not reniform. The bowel is usually in front of the mass, although in tumors of the right kidney the cecum and colon may be pushed to the inner side, and in tumors of the left kidney the colon may be pressed outward.

The disease of the kidney attended with enlargement are: malignant tumors, cysts of the kidney, hydronephrosis and pyonephrosis, abscess, and perinephritic abscess. The kidney is also palpable when movable.

Circulatory Derangements.—Congestion of the Kidneys.—The causes of *acute congestion* of the kidney are practically those of acute nephritis. It occurs characteristically at the onset of this condition, and it is impossible to clinically differentiate the two derangements except by their subsequent course.

Chronic congestion of the kidneys is usually a part of general venous stasis; valvular heart disease with secondary dilatation and pulmonary emphysema often give rise to the condition. Obstruction of the renal veins, as by tumor, is a much less frequent cause. *Urine:* the quantity is diminished, the color dark, the specific gravity high—1.020 to 1.030. Uric acid and urates are present in excess. Albumin is not present at first, but soon makes its appearance at intervals and in small quantities, later becoming constant and more abundant. The sediment contains hyaline casts and a few red blood cells.

The condition is recognized by its association with congestion in other organs; by the diminution in the amount of urine, its high specific gravity, and excess of uric acid and urates. This form of congestion is serious because it leads to chronic nephritis.

Thrombosis and Embolism.—Primary thrombosis is extremely uncommon, embolism more common. The latter is recognized by the sudden appearance of renal pain, tenderness of the kidney, oliguria, and hematuria. A septic embolus causes chills, fevers, sweats, and other manifestations of sepsis.

Inflammations.—The classification of the inflammations of kidneys is extremely difficult, as not only clinically are the symptoms of the various forms of nephritis intermingled, interchanged, and intimately correlated, but histologically morbid kidneys vary so that

pathologists have not been able to satisfactorily agree upon the differentiation of the various types of nephritides; while even more difficult is the coördination of the clinical picture with the postmortem findings. In any form of acute nephritis the involvement is diffused throughout the whole of the renal tissues and the clinical manifestations of the disease are essentially the same, no matter whether the pathological changes have involved primarily and chiefly the parenchymatous or the interstitial tissue (excepting of course an acute suppurative nephritis). In chronic nephritis, however, a sharp clinical and histological division can be made between a form of nephritis which follows the subsidence of an acute attack or begins insidiously, and which involves chiefly the parenchyma (chronic parenchymatous nephritis) and a form in which the lesions are largely in the interstitial tissue and are merely a part of a widespread change in the vascular system throughout the entire body (chronic interstitial nephritis).

Acute Nephritis (*Acute Parenchymatous Nephritis; Acute Diffuse Nephritis; Acute Bright's Disease*).—*Causes*.—There may be merely a history of exposure to cold. At times the disease seems to occur without any definite cause. It is a frequent complication or sequel of the infectious diseases, notably scarlet fever, diphtheria, pneumonia, influenza, smallpox, erysipelas, and typhoid fever. Certain autogenous metabolic poisons, elaborated in the course of pregnancy, anemia, diabetes, gout, and as a result of severe burns, or exogenous poisons, as turpentine, cantharides, phenol, spices, alcohol, ether, or chloroform acting upon the kidney may be the etiological factors.

Symptoms.—The course of the disease may be mild, presenting only the urinary changes, or there may be, in addition, local and general symptoms.

In mild cases the urine is diminished in amount, the color is dark, the specific gravity usually high, micturition is frequent. There is a small amount of albumin, with a few epithelial and blood casts, and sometimes blood. At the termination of the disease, hyaline casts are found. In severe cases the disease is often ushered in by chill, attended and followed by pain in the loins, with fever, headache, and restlessness. The urine contains a large amount of albumin and an abundance of hyaline, granular, epithelial, and blood casts, and free red and white blood cells. The fever, if it continues, is moderate and irregular in type; the pain in the loins is sometimes very severe. In the first forty-eight hours the characteristic symptoms that follow the chill and accompany the urinary changes are headache, sleeplessness, more or less profound stupor, muscular twitchings, and general convulsions. Eye symptoms may be present. Instead of cerebral symptoms there may be marked dyspnea, and with both, nausea and vomiting are of frequent occurrence. The heart's action is increased in force, and at first the frequency is often lessened, later increased. The left ventricle may become hypertrophied after five or six weeks, and dilatation may rapidly succeed it. The aortic second sound is accent-

uated. The pulse is hard, and exhibits the characteristic features of high tension.

Dropsy or edema is one of the most constant symptoms. It appears first in the face, especially the eyelids, and may be limited to this region, or it may extend to the lower extremities and to the scrotum. It may progress to anasarca, and one or more of the serous cavities fill up with fluid. Edema of the mucous membranes, as the conjunctiva, the soft palate, and the glottis, occur at times.

Dyspnea may be a pronounced symptom, due to uremia (uremic asthma), edema of the glottis, effusion into the pleura, or bronchitis. If dilatation of the heart occurs, the ensuing dyspnea may be a direct result or due to the secondary edema of the lungs.

With or without the occurrence of nausea or vomiting, there is always loss of appetite, and the bowels are usually constipated.

Prostration is common; often there is emaciation, and frequently a severe secondary anemia develops. Symptoms of uremia may occur at any time.

Diagnosis.—Acute nephritis is diagnosticated largely by the examination of the urine. Etiological associations are of value. There may be no symptoms until the onset of uremia. In some instances the disease resembles typhoid fever. The albuminuria and cylindruria that arise during the course of acute infectious diseases must not be considered as acute nephritis unless erythrocytes, much albumin, and many casts appear in the urine and some of the general symptoms of nephritis develop.

An acute exacerbation of a chronic nephritis may simulate an acute primary nephritis, but a careful history will usually disclose past symptoms which are suggestive of a previously existing nephritis. The possibility of vascular changes in the kidney being the predominant pathological picture (*acute glomerular nephritis*) is suggested by the fact that such changes occur most frequently in postscarlatinal nephritis. The urine is of a high specific gravity, and contains much albumin and many erythrocytes; dropsy, anuria, and uremia are pronounced.

Chronic Parenchymatous Nephritis (*Chronic Diffuse Nephritis*).—*Causes.*—This form usually follows acute nephritis. It develops in the course of syphilis, tuberculosis, malaria, endocarditis, disease of the bone, and prolonged suppuration. Frequent exposure to cold and wet, residence in damp dwellings, and the alcohol habit are causal conditions. It usually occurs in middle life, more frequently in men; but as a secondary disease usually attacks young adults after scarlatina.

SYMPTOMS.—The disease succeeds an acute nephritis or more frequently it develops slowly. General symptoms may be first observed. Dropsy may develop at first and continue throughout the disease recur at long intervals. The appearance of the patient is striking. The skin is of a peculiar pallor and pasty in appearance. The sclerotics are very white. The anemia which gives rise to the pallor is very marked, and often closely resembles that of pernicious anemia. Head

and sleeplessness are common symptoms. Pronounced acute uremia does not often occur. Chronic uremia may prove fatal by the patient lapsing into a typhoid state in which delirium alternates with stupor.

Albuminuric neuroretinitis frequently develops in the course of the disease.

Dyspnea is often observed, and may be due to any one of the many causes previously described as producing this symptom in the course of nephritis. It is frequently limited to sudden attacks which develop in the night or early morning. There is often some bronchial catarrh. Nausea and vomiting are common symptoms. The appetite is lost. Hypertrophy of the left ventricle takes place in many cases. The pulse is generally of high tension and arteriosclerosis often supervenes. Symptoms such as headache and vertigo arise on account of the profound anemia as well as the toxemia.

Urine.—It must not be forgotten that the course of the disease and the urinary symptoms are often quite variable in chronic nephritis. The urine may be normal in amount, but during the exacerbations it is scanty or suppressed. In the early cases, the specific gravity varies between 1.018 and 1.025 or higher, the extent of the increase depending upon the amount of urine excreted, the smaller the quantity of urine, the higher the specific gravity. In the later stages the amount of the urine may be increased and the specific gravity is decreased. Albumin is present in large amounts. When the disease is most active and the dropsy at its height, the quantity of albumin is very large. In the quiescent period of the disease the amount is lessened. Epithelial, fatty, and granular casts are abundant; red blood cells are often found.

COURSE.—The disease is characterized by remissions and exacerbations. During the exacerbations any one of the prominent symptoms that occur in renal inflammations may be present. Edema is the one symptom which occurs most frequently, and is likely to continue the longest. The further course of the disease may be as follows: (1) The symptoms may be continuous and progressive in severity, death taking place at the end of one or two years. (2) The symptoms may continue for several months and the patient finally improve. Recurrent attacks occur, the symptoms being more severe with each attack. (3) The patient may apparently recover, but the urine continues to be of low specific gravity, and contains some albumin. (4) The symptoms may persist in a mild degree for years. (5) Chronic interstitial nephritis may supervene.

DIAGNOSIS.—Though suggested by the cardinal symptoms of edema and anemia, the diagnosis must be based upon the urinary examination.

Chronic Interstitial Nephritis (*Chronic Diffuse Indurative Nephritis*).

—*Causes.*—This form of nephritis follows chronic congestion of the kidney and chronic parenchymatous nephritis. It is also caused by alcohol, lead, gout, syphilis, malaria, and by the factors that cause arteriosclerosis. The arteriosclerotic form is associated with other phenomena which develop as a result of the endarteritis, *e. g.*, hyper-

trophy of the heart, and to which the term cardiovascular-renal disease is frequently applied. Overeating, worry, insufficient exercise, and the wear and tear and stress of modern life are etiological factors of importance. In a certain number of cases the lesions appear to be solely the result of senility. This form of nephritis is notably prevalent in several generations of a family.

SYMPTOMS.—The onset of the disease usually occurs late in life, although well-defined cases may occur in early life. The progress at first is very insidious, and the disease may have advanced to an extreme stage without the occurrence of a single symptom. Death, indeed, may be due to other causes; or a person in apparently perfect health may suddenly manifest symptoms of uremia, or may develop apoplexy or some other usual accompaniment of interstitial nephritis. With the exception of the state of the urine, the only symptom present may be loss of flesh and strength. At the same time the skin becomes dry and harsh. Edema, however, is not usually present unless there is dilatation of the heart.

Uremic symptoms may occur at any time in the course of the disease. They may occur suddenly or develop very gradually. Headache is most common and constant. (See Subjective Diagnosis—Uremia.)

Pulmonary symptoms other than those due to uremia may be caused by an intercurrent bronchitis, pneumonia, or pleurisy. Chronic bronchitis or edema of the lungs may be present. Emphysema often occurs, while hydrothorax is found late in the disease.

There is always a tendency to chronic inflammation of the mucous membranes and to acute inflammation of serous membranes in the course of chronic diffuse nephritis.

Gastro-intestinal Symptoms.—The most common gastro-intestinal symptoms are the occurrence of morning nausea or of morning vomiting; the occurrence of spasmodic vomiting at irregular periods, or the occurrence of violent, acute vomiting, which is followed in two or three days by other symptoms of uremia. The bowels are usually constipated. The onset of uremia may be characterized by violent and profuse serous purging. Cirrhosis of the liver, chronic gastritis, and chronic enteritis are frequent complications appearing in the course of the disease.

Neuroretinitis is a frequent complication of nephritis, and may advance more rapidly than other complications, so that dimness of vision, blindness, or other eye symptoms may cause the patient to consult an oculist before attention is called to the condition of the kidneys.

Cardiovascular Symptoms.—These are constant. The left ventricular hypertrophies. The aortic second sound is accentuated. There is high tension. The arteries become more prominent and show all the signs of endarteritis. In the later stages, as nutrition fails, dilatation of the heart takes place with insufficiency of the valves, and the development of a train of symptoms due to these changes. On account of the atheroma, and aided by the hypertrophies

rupture of the vessels frequently takes place. Apoplexy is, therefore, of common occurrence, and hemorrhage into other organs sometimes occurs. The renal disease is often not suspected until after the patient has had an attack of apoplexy.

Urine.—The quantity is increased, the color clear, and the specific gravity low. The albumin is small in amount, or may be absent. Repeated examinations extending over a considerable period of time may disclose its presence. Hyaline casts are present in small numbers. In some cases it may be necessary to examine a dozen or fifteen slides before they are found. Rarely the urine is bloody at irregular periods in the course of the disease, or actual hematuria may take place.

DIAGNOSIS.—This is based upon the association of hypertrophy of the heart, arteriosclerosis, high blood-pressure, and the excretion of large amounts of urine of a low specific gravity containing but traces of albumin and occasional hyaline casts. Several clinical manifestations of interstitial nephritis are observed. In the latent form the disease may have advanced to an extreme degree without any symptoms of renal disease during life, with death taking place from an intercurrent disease or accident. The patient enjoys good health until the occurrence of pneumonia or inflammation of a serous membrane. Often the local inflammatory symptoms are slight or masked by the symptoms of renal disease, which develops rapidly.

In another group of cases some special symptom only may be complained of. In some instances it may be gastric catarrh, in some eye symptoms alone may be present, while in others hemicrania or other forms of headache are observed. With the headache there is usually vomiting. Again we may have constant neuralgia or persistent muscular rheumatism as the only symptom. Nose-bleed is a symptom which may be the only indication of chronic nephritis, particularly if the epistaxis occurs frequently.

In other cases the course is not latent, but characterized by a series of attacks of varying intervals. During the attacks the symptoms resemble those of the acute form of nephritis with acute uremia. The tension of the arteries is higher and the urine contains albumin, and is of low specific gravity during the attack; during the interval albumin is found at irregular times. It is to be remembered that chronic interstitial and chronic parenchymatous nephritis frequently affect the kidney at the same time. In such a case the clinical symptoms of one type usually are more pronounced than those of the other, though frequently it is impossible to say which is the primary and which is the active type of chronic nephritis.

Suppurative Nephritis (*Abscess of the Kidney*).—Pyogenic bacteria are conveyed to the kidney either through the blood, as in pyemia and ulcerative endocarditis, or by the ureters, as when it follows pyelitis or cystitis, or by contiguity from near-by inflammations.

Symptoms.—The symptoms are those of the primary disease, and the affection is usually recognized only postmortem. Or the symptoms

are merely those of suppuration. Pus is seen in the urine only on rupture of the abscess into the pelvis of the kidney. There is frequently tenderness over the affected kidney.

Tuberculous Nephritis.—Fever, emaciation, anemia, and prostration characterize the course of the disease. Tuberculosis is usually found elsewhere. There may be no other symptoms. Sometimes hydro-nephrosis or a tumor is present which may be in the loins, or may be in front, above, and a few inches to the right or left of the umbilicus. The urine is normal or contains pus and detritus, or even bacilli. The finding of the latter is often necessary to establish a diagnosis. In all instances of pyuria, renal tuberculosis should be suspected. The testicles, bladder, and prostate should be carefully examined for primary tuberculosis.

Tuberculosis of the kidney presents symptoms like those of pyelitis, renal calculus, or a new growth. It is almost impossible to distinguish any one of the four until an interval has elapsed. In all cases the patient suffers from dull pain, sometimes with a bearing-down sensation. Hematuria occurs, and the patient is liable to attacks of renal colic. These symptoms may continue until a tumor can be made out. Even before this, pain, which may extend all along the urinary tract, will be elicited on palpation. With the occurrence of the tumor the general symptoms of tuberculosis arise.

Malignant Tumors of the Kidney.—The primary malignant tumors of the kidney include hypernephroma, the result of growth of misplaced adrenal tissue, sarcoma, and carcinoma. The first variety of malignant tumor is the commonest form affecting adults. Sarcoma of the kidney is comparatively frequent in childhood, but carcinoma is an extremely rare condition. Secondary malignant tumors may develop in the kidney late in the course of malignant disease elsewhere.

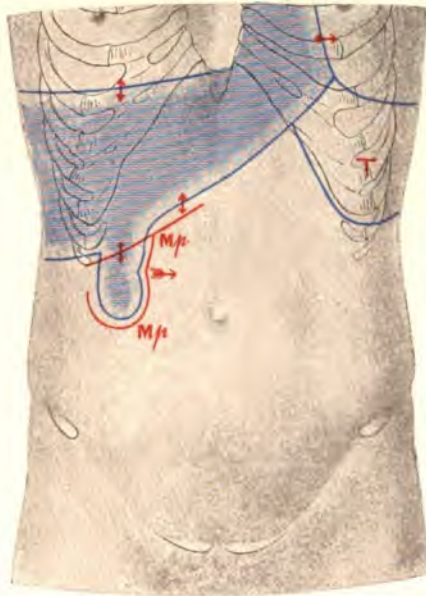
The benign tumors of the kidney are of little clinical interest.

Symptoms.—In life the disease may advance considerably before it presents any signs. If symptoms are complained of, they are usually limited to pain, the occurrence of hematuria, or the development of a tumor. The pain is dull and seated in the lumbar region. It may be neuralgic in character, and indeed, there may be true sciatica from pressure of the tumor. The hematuria may be constant or intermittent, and is a valuable symptom. The tumor is firm; its surface is smooth or nodulated. It may be felt in the loins, and in front above the umbilicus, a few inches to the right or left of the median line; the descending colon lies in front of the tumor. The latter may grow with great rapidity and attain an enormous size, filling the abdominal cavity and giving rise to pressure symptoms in surrounding organs. It is movable either by the hands or with respiration. On percussion the resistance is increased and the note is dull, except in front, where the colon gives a tympanitic note. Rare physical signs are pulsation and a blowing murmur.

The general symptoms are those of cachexia. Symptomatic varicocele

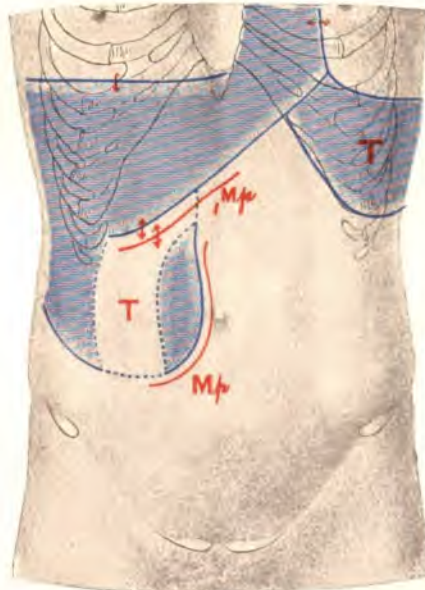
PLATE XXVII

FIG. 1



Movable Kidney.

FIG. 2



Sarcoma of the Right Kidney.

may occur. The examination of the urine, save that it discloses the presence of blood, is negative. In this sense it is of value. Pus occurs if there is secondary infection or if calculi precede the growth. Rarely fragments of tumor tissue are said to be detected. In order to determine which kidney is affected, the urine should be obtained separately from each organ by ureteral catheterization.

Diagnosis.—The occurrence of a retroperitoneal growth associated with hematuria is the important diagnostic syndrome. A malignant renal tumor is distinguished from other enlargements of the kidney by the presence of anemia and cachexia and absence of fever. Retroperitoneal sarcoma is differentiated by its more central position and greater fixation. Ovarian tumors, and less frequently tumors of the uterus, spleen, and liver may simulate renal new growths, but careful examination will usually demonstrate the true origin of the tumor.

Cystic Kidneys.—1. **Congenital.**—The kidney consists of a small mass of cysts filled with clear fluid. It may at birth interfere with delivery, on account of its large size, or may not be discovered until adult life. The urine resembles that of chronic interstitial nephritis. The chief symptoms are intermittent hematuria, bilateral kidney tumors, and the manifestations of chronic interstitial nephritis.

2. **Acquired.**—These include (a) multiple small cysts due to obstruction of renal tubules; (b) large solitary cysts probably due to the same cause; (c) parasitic or echinococcus cysts.

The first two forms are usually symptomless and are discovered at autopsy. The latter form causes enlargement of the kidney, hematuria, and pyuria. If the cyst ruptures into the pelvis, hooklets and shreds of membrane may be found in the urine.

Hydronephrosis.—This consists in a dilatation of the pelvis of the kidney with urine, which is prevented from escaping by obstruction of the ureter. It may be congenital; it may be caused by obstruction of a ureter, as, by a stone, or an inflammatory substance; by the pressure of a tumor; and by kinking of the ureter, as in movable kidney.

Symptoms.—In addition to the symptoms of the causal condition we have, upon the development of hydronephrosis, the presence of a tumor arising in the region of the kidney and extending toward the middle line. Sometimes fluctuation can be detected; often it cannot. Variations in the size of the tumor may occur with changes in the amount of urine passed, often suddenly disappearing entirely after a copious discharge of urine. When on one side, the urine may be normal; when on both sides, it is diminished; anuria and uremia may occur. If pyelitis is present, pyuria is observed. Pain may or may not be present. Gastric symptoms are very common. Either constipation or diarrhea is seen. Hypertrophy of the left ventricle may occur, as in chronic nephritis.

Intermittent hydronephrosis is associated with movable kidney. It is characterized by the development of a renal tumor which appears at varying intervals, and by pain, nausea, and vomiting; during this period

Perinephritic Abscess.—Inflammation of the capsule of the kidney and the surrounding fibrous tissue arises usually from extension of inflammation and suppuration from the kidney, but may be the result of injury or of extension of inflammation from a diseased appendix, a carious spine or an empyema or follow one of the infectious diseases (pyemic).

Symptoms.—Chills and fever, pain, difficulty in defecation are present. The general condition suffers. Finally, in all cases a swelling, hard at first, develops in the lumbar region; then edema of the skin follows and fluctuation is detected. The abscess may descend and point above Poupart's ligament. It may press upward and cause dyspnea. Great tenderness and pain in the region of the swelling may arise, and the pain may radiate to the leg. Irregular septic fever and chills appear. The urine is not generally changed unless there is some communication with the pelvis or ureter. The patient lies on his back turned toward the affected side. The knee and hip of this side are flexed, and the thigh is rotated outward. The affection may simulate coxitis and appendicitis. The usual signs of confined suppuration exist, and pulmonary or pleural complications may occur. As the abscess progresses, the local signs of suppuration become more marked, the skin reddens, and pus may be discharged externally.

The most marked subjective symptom is pain, which may amount to agony and which is paroxysmal; soreness from restricted motion of the psoas muscle is apt to be complained of.

Nephrolithiasis (Renal Calculus).—Renal calculi vary in size from minute particles of "sand" through "gravel" to "stones." The last may be from the size of a cherry stone to one large enough to fill the pelvis of the kidney. They consist largely of calcium oxalate, and are hard, pale yellow to deep brown, and crystalline; the larger ones are arranged in distinct layers. More rarely we have calculi composed largely of uric acid and the urates which, however, are present in small quantities in all stones. Some stones have alternate layers of the two salts; others consist of phosphates, but usually the centre is of uric acid or calcium oxalate, the phosphates having been deposited after the urine becomes alkaline. Very rare forms are composed of cystin, xanthin, indigo, etc. The shape, appearance, and consistency seem to depend upon the rate and process of formation rather than upon the chemical composition of the stone (Kahn).

Nephrolithiasis is a common affection. It is a disease of the middle and upper classes. This is particularly true of the uric acid calculus. There does not seem to be much difference between the two sexes in regard to frequency. Sedentary occupation and an indoor life are predisposing factors.

Symptoms.—With the exception that some present no symptoms whatever, the cases may be divided into three groups:

(a) Calculi may remain in the pelvis of the kidney, and not cause any renal symptoms. They may cause gastric disturbance or catarrh

A turn indicate the nature
~~specific organisms~~

Myositis (*Inflammation of Muscles*).—In *suppurative myositis*, secondary to a pyemia, there are pain, swelling, and loss of power, the condition going on to abscess formation. *Syphilis* may cause either a cellular myositis early in the disease or, later, gummata may form. *Primary myositis* is rare. It may occasionally be seen as a *dermatomyositis* in which the inflammation begins in the lower extremities and gradually ascending involves other muscles of the body. They are swollen, hard, and painful on pressure. Local edema of the skin over the muscles is seen associated with a dermatitis. Occasionally hemorrhage into the involved muscles may occur (hemorrhagic polymyositis). Atrophy supervenes in groups of muscles. Death ensues when the respiratory muscles are involved. The disease must not be confounded with trichinosis.

Myositis ossificans is a rare disease of the muscles. The muscle tissues undergo gradual ossification, either in localized spots or progressively in widespread areas. The disease lasts many years.

Heat-cramp (*Edsall's Disease*).—Pain and cramps of the muscles, occurring in those who work in extreme heat, and who do much muscular work at the same time, is a rare and until recently an unrecognized condition. The attack lasts about twenty-four hours and is described as continuous fibrillary contractions of the muscles, particularly the calf muscles, associated with paroxysmal, painful, tonic spasms of the muscles of the forearms, legs, and abdomen, occurring spontaneously and excited by voluntary movement.

Myotonia Congenita (*Thomsen's Disease*).—This is an hereditary disease, and may occur in several generations of a family. The disease begins in childhood. Tonic cramps occur in the muscles when voluntary movements are attempted, particularly noticeable in walking. The muscles become rigid and fixed when put in action. The rigidity soon wears off and the limb can then be used. Though both the arms and the legs are affected, the patients are usually well-nourished. There are no atrophies. The muscles are irritable, so that mechanical stimulus or pressure causes tonic contraction. Movement and cold aggravate it. Sensation and the reflexes are not affected and there is no evidence of disease of the cerebrospinal system. The myotonic reaction described by Erb is induced. (See page 200.)

Paramyoclonus Multiplex.—In this affection there is clonic, constant, or paroxysmal contraction of the muscles, usually confined to the extremities. The contractions are bilateral and rhythmical, as if from repeated electric shock. Tremor of the muscles may be present in the intervals.

Myasthenia Gravis (*Asthenic Bulbar Palsy*).—A rare disease of the muscles characterized by rapid exhaustion of the muscles upon slightest exertion and subsequent recovery from the fatigue if the exertion is not persisted in. The muscles innervated by the bulb are first affected, but later other muscles are also involved. The muscles show an absence of fibrillary twitching and atrophy, and the presence of the

even during sleep, the lips remain apart, and saliva dribbles from them. Ultimately, the other muscles of the body are involved, and the disease assumes the characteristic of the preceding form.

Pseudohypertrophic type commences in early life—from the third to the sixth year. Ordinarily the muscles of the calves are first involved. These become greatly enlarged, hard, and there is great loss of power. Other muscles of the legs are next involved, then those of the back, and perhaps the arms. Not all the muscles that undergo atrophy show a preliminary hypertrophy. The electrical reactions remain normal, and the loss of power is due merely to the atrophy of the true muscle substance. The gait is waddling, and the patient is unable to arise from the ground except by getting upon the hands and knees and then gradually climbing up his legs. There is usually lordosis or scoliosis, and occasionally contractures occur, leading to formation of club-foot. Sensation remains unimpaired throughout the disease. The reflexes show diminution corresponding to the loss of muscular substance. The intelligence of the patient in this, as in the two preceding forms, remains intact. The course is slowly progressive. Similar cases without pseudohypertrophy are sometimes called the *Leyden-Moebius form*.

(For discussions of paralysis, spasm, tremor, contraction, etc., see Examination of the Nervous System.)

sacro-iliac disease or relaxation of the joint ligaments, or to pressure by tumors.

Coccygodynia.—A neuralgia of the coccygeal plexus which occurs most often in women. A misplaced or fractured coccyx may be the real cause of the pain.

Neuralgia of the Feet.—This includes metatarsalgia (Morton's disease), pododynia (painful heel), and plantar neuralgia (tender toes).

Visceral Neuralgias.—Neuralgias, such as gastralgia, hepatalgia, and nephralgia, may occur, but the neuralgiæ-like pain is probably due to some anatomical cause.

Meralgia Paresthetica.—This is a neuralgia (or neuritis) in the area of the distribution of the external cutaneous nerve of the thigh, usually unequally bilateral, and made worse by prolonged exercise, either walking or standing. Frequently there is a tender point just below the anterior superior spine of the ilium. Sensory disturbances in the form of hypesthesia and hyperalgesia are common.

Neuritis.—Inflammation of a nerve may be confined to a single nerve (localized) or it may involve many nerves (multiple or polyneuritis). *Localized neuritis* is characterized by pain, localized in the course and distribution of the nerve affected, paresthesia, tenderness, and perhaps paresis or paralysis of groups of muscles. The pain is made more severe if the limb is held in such a position that the nerve is stretched. The nerve may be distinctly enlarged to touch. Often the disease is progressive, extending from the peripheral to the more central nerve trunks. This is spoken of as ascending neuritis. Along the course of the nerve there are often vasomotor and secretory disturbances, such as pallor, redness, edema, or the lesions may be more severe, such as atrophy of the skin, with glossiness of the skin, or trophic changes in the hair and nails. The symptoms vary according as the nerve affected is chiefly motor or chiefly sensory. *Inflammation of a motor nerve* causes flaccid paralysis in the group of muscles supplied by the nerve, and if the inflammatory process persists, these muscles rapidly atrophy and give the reactions of degeneration. The reflexes in the parts controlled by these muscles are lost. The diagnosis can usually be made by a careful study of the distribution of the paralysis. The paralysis may be permanent, or recovery may ensue if the process has not proceeded too far. *Inflammation of a sensory nerve*, in addition to pain, is usually characterized in the early stages by hyperesthesia of the skin in the distribution of the nerve, which later becomes anesthetic, and is often associated with hyperalgesia (anesthesia dolorosa). Vasomotor and trophic changes are common.

Multiple neuritis is characterized by the appearance of the symptoms of the disease in a number of nerves at the same time. The nerves of the limbs are far more frequently affected than those of the trunk. The symptoms are modified by the cause. In the early stages there are usually slight paresthesias of the limbs, and later on, weakness of the muscles, particularly the extensors, and

to foot-drop and wrist-drop. The disease usually affects all four extremities, and phenomena similar to those of a localized neuritis, appear in the affected parts. In lead-poisoning the disease is sometimes unilateral, is usually restricted to the arms, and the sensory disturbances are very slight or absent. There is paralysis of the extensor muscles of the arm, which, in severe cases, goes on to muscular degeneration. Neuritis may also be produced by arsenic. Diphtheritic polyneuritis is usually characterized by paralysis of the muscles of the palate, causing regurgitation of food through the nose, but occasionally the muscles of the limbs are also involved. In certain of the chronic forms of polyneuritis, instead of, or with, the loss of power, there is marked loss of coördination and ataxia.

Infectious multiple neuritis may be due to the direct action of the organisms upon the nerves, as in beriberi, malarial and leprous neuritis, or to the toxin elaborated by an organism, as in diphtheria, smallpox, typhoid fever, and septicemia.

Herpes Zoster.—This is probably an acute specific infectious disease, and is characterized by severe pain and a herpetic eruption along the course of a sensory nerve. The onset is usually sudden, with fever, pain, and in three or four days the vesicular eruption which appears along the course of the affected nerve, most frequently one of the intercostal nerves of one side. At the end of seven to ten days the vesicles dry up and the pain disappears.

Neuromas.—These may be true tumors of nerve fibers or false tumors situated on nerve fibers. The latter include (1) fibromata, sarcomata, or myomata; (2) tubercula dolorosa, multiple painful tumors involving the cutaneous sensory nerves of the face, breast, or about the joints; (3) plexiform neuromas, multiple tumors made up largely of fibrous tissue; (4) amputation neuromata, painful enlargements of divided nerves. These tumors may only be recognized by inspection and palpation or they may cause paresthesias, anesthetics, or paralysis.

Diseases of the Cranial Nerves.—These are considered either in the sections on the Examination of the Organs of Special Senses or in the section devoted to the Examination of the Nervous System.

Diseases of the Spinal Nerves.—The Cervical Plexus.—Paralysis of one or both *phrenic nerves* causes unilateral or bilateral paralysis respectively of the diaphragm, shown by immobility or retraction of the upper abdomen during inspiration, bulging during expiration, and dyspnea upon exertion.

The Brachial Plexus.—Lesions damaging the whole or part of the plexus cause paralysis of the nerves arising from the injured roots. There may be paralysis of the whole arm and some of the shoulder muscles if the whole plexus is involved; paralysis of the upper arm in lesions of fifth or sixth roots, and paralysis of the lower arm in lesions of the seventh and eighth cervical and first thoracic roots. Lesions of the following individual nerves of the plexus cause the following main conditions: (1) The *long or posterior thoracic nerve*—impairment of the shoulder

hands. Later, there may be spastic paraplegia of the legs, with anesthesia of the body below the affected segment.

Spinal Leptomeningitis.—Acute leptomeningitis is characterized by intense pain in the back, radiating into the legs; rigidity of the spinal column, with opisthotonos; intense hyperesthesia of the skin of the body, and, if the disease lasts long enough, paralysis. Kernig's symptom, that is the inability to extend the flexed leg as a result of flexor cramp, is present. It is always due to an infection, and, therefore, fever, chills, and prostration are present. The diagnosis is greatly assisted by examination of the fluid withdrawn by spinal puncture (*q. v.*).

Chronic leptomeningitis is quite uncommon. It may be due to syphilis or tuberculosis.

Spinal Meningeal Syphilis.—This usually causes a meningomyelitis in which both membranes are affected and which may produce a great variety of symptoms. They are: pains due to pressure upon the posterior roots, girdle pains of the body, and occasionally paralysis of the muscles of the extremities, with atrophy and degeneration. Often, as the spinal cord is also involved, the symptoms of pressure appear or transverse myelitis (*q. v.*) or Brown-Séquard syndrome (*q. v.*). The sensory symptoms consist of hyperesthesia, hypesthesia, or anesthesia. The tendon reflexes of the lower extremities may be lost and reappear, and by some this is supposed to be pathognomonic of the disease.

Hemorrhage into the Spinal Membranes.—The symptoms which usually appear suddenly are those of pressure on the cord in the region in which the hemorrhage takes place.

Acute Diseases of the Spinal Cord.—**Hemorrhage into the Cord** (*Spinal Apoplexy*).—This is characterized by the sudden interruption of the function of the cord at a certain level. There is usually, at the time the hemorrhage occurs, severe pain, then rapidly developing paralysis of the legs, which may be flaccid if the lumbar region is involved, or spastic if the lesion is higher up. Hematomyelia into the cervical region may cause paralysis of the arms, but death usually occurs suddenly. The sensory disturbances are irregular in character. At times there is dissociation of sensation, more frequently complete anesthesia up to the level of the hemorrhage; there is interference with the functions of the bladder and rectum. Occasionally the hemorrhage involves particularly one side of the cord, or only one-half of the gray matter, producing the syndrome of Brown-Séquard (*q. v.*). The diagnosis can frequently be made from the subsequent course of the case. If death does not occur, rapid improvement is usually the rule. The sphincters regain their functions, power returns to the limbs, and ultimately the patient may recover completely. In some cases, however, recovery, although pronounced, is only partial.

Caisson Disease.—The disease attacks workers in caissons where the air is under high pressure. It is due to the sudden expansion of gases in soft tissues (brain and cord) held within inexpandible tissue (bone). The symptoms appear a short time after the return to normal

atmospheric pressure. There is usually epigastric pain and vomiting, followed by severe pains in the joints, the back, and lumbar regions. Paraplegia or hemiplegia soon follows. In severe cases the bladder and rectum are paralyzed. Cerebral symptoms, in severe and fatal cases, include headache and vertigo, unconsciousness and coma.

Myelitis.—Inflammation of the spinal cord may be acute in onset; subacute, the symptoms developing in from three to six weeks; or chronic, a longer period being required for the full development of the symptoms. The disease may be diffuse, focal, disseminated, or transverse, according to whether the lesion affects an extensive area, a small localized area, more than one focal point or the whole thickness of the cord.

The *acute form* is associated with constitutional disturbances, that is, chills, fever, and malaise, and is occasionally ushered in with a convulsion. Ordinarily the dorsal part of the cord is affected; and there are in the earlier stages weakness and paresthesia of the legs, and perhaps a girdle sensation and hypesthesia over the spine, in the zone supplied by the involved segment. Not infrequently painless spasms occur in the legs. In the course of a few days or hours the weakness of the legs increases until there is complete paraplegia. The tone of the muscles is enormously exaggerated, the knee-jerks are increased, and there are patellar and ankle-clonus, and Babinski's sign. The limbs are usually spastic. From time to time the muscles give violent twitches. At first there is hypesthesia, soon passing into complete anesthesia up to the segmental level, at which point there is girdle sensation, and above it there is a zone of hyperesthesia. The muscles supplied by the affected segment atrophy and give reactions of degeneration. Those in the region below maintain their nutrition for a considerable time. There is difficulty in micturition, and finally overflow from retention. The urine becomes alkaline, cystitis develops very rapidly, and is often followed by extensive sloughing of the surrounding parts. Bed-sores occur early and extend deeply. Trophic lesions also occur in the legs, the skin becomes thin and glazed, and the toe nails are brittle. After the acute stage has passed more or less improvement may occur, characterized by gradual return of power in the legs and partial recovery of sensation.

The symptoms vary from the above typical picture of a dorsal lesion, according to the extent and seat of the disease. Sensory or motor symptoms may predominate. A monoplegia or irregular paralysis, and irregular areas of anesthesia may be noted. A lesion in the cervical region causes symptoms similar to a thoracic lesion plus involvement of the arms, shoulders, and at times the muscles of the neck. A lesion causes a paraplegia with pains in the leg, atrophy of the muscles, paralysis of the sphincters of the bladder and rectum and, most important, absence of the deep reflexes.

Chronic myelitis is a term which may be applied either to the condition after the subsidence of the acute symptoms or to the syndrome when they appear slowly and insidiously.

Acute Ascending Myelitis.—Landry's Paralysis.—This is characterized by progressive paralysis of the legs, arms, and muscles of the throat, leading ultimately to death. The prodromes consist of slight fever, malaise, and paresthesia in the legs. These are followed by weakness of the legs, which may involve both, or at first only one. This gradually ascends, and at the same time the paresthesia becomes more distressing. There are few or no objective sensory disturbances. The reflexes are lost, and the paralysis is flaccid. The paralysis gradually ascends, and when the thorax is involved, the patient usually has rapid respiration and complains of dyspnea. Later there are symptoms of bulbar involvement, as difficulty in deglutition. The intelligence remains normal, there is never loss of consciousness, and there is no disturbance of the function of the bladder or rectum.

Anterior Poliomyelitis.—(See page 502.)

Chronic Diseases of the Spinal Cord.—Tabes Dorsalis (*Locomotor Ataxia, Posterior Spinal Sclerosis*).—Tabes usually begins in the decennium from thirty to forty. It affects men more commonly than women, and is exceedingly rare among negroes. In from 95 to 97 per cent. of cases tabes follows a syphilitic infection, occurring from five to twenty years after the initial lesion. It is divided clinically into three stages: the preatactic, the atactic, and the paralytic. The symptoms of the *preatactic* stage frequently commence with disturbance in the nerves controlling the movements of the eyeball. There may be paresis of the abducens, giving rise to diplopia; of the levator palpebræ, giving rise to ptosis—both of which may be transient, and may be discovered only by the history of diplopia; or drooping of the eyelids. The reaction to light on the part of the pupil may be sluggish or absent; while the reaction to accommodation still persists (Argyll-Robertson pupil). The symptoms in the nerves of the lower extremities are particularly the lancinating pains that are felt in the posterior portion of the thigh. These are paroxysmal, and the patient feels as if he had been stabbed. They are at times merely a dull ache, and are often confused with rheumatism. These *pains* may persist for years as an isolated symptom. The knee-jerk is absent (*Westphal's symptom*), and the patient may note that it is somewhat more difficult to walk in the dark. Often there is distinct weakness in the legs. The station in the early stage is usually only slightly affected. There is a sense of constriction about the body (girdle pain), and sometimes hypæsthesia of the lower extremities that may be associated with a slight hyperalgesia in the zone just above it. The patient may also remark that they have slight difficulty in urination and some diminution of sexual potency. The second stage, or the stage of *ataxia*, is characterized by the symptoms of the preceding stage, all of which are now pronounced. In addition the patient exhibits incoördination of movement, especially in the lower limbs. Station is so impaired that it is usually impossible for him to stand alone with the eyes closed and the feet together (*Romberg's symptom*). Walking in the dark is difficult and

results in frequent falls. In the daylight, the patient can usually walk quite well, but lifts the feet higher than usual from the ground, and separates them widely. (See Ataxic Gait.) The incoördination is manifested by the difficulty with which the patient performs certain purposeful movements. There is absolute loss of the tendon reflexes, even when reinforced. The nutrition of the muscles remains good, and the electrical reactions are not altered. There are paresthesias, especially in the lower extremities (a sensation as if walking on cotton is often noted), analgesia in the same situation, or sometimes delay in the conduction of pain. Visceral anesthesia is also present. The skin reflexes are often preserved late in the course of the disease. Micturition is sometimes difficult; at other times there is incontinence, but insufficiency of the sphincter ani rarely occurs. Impotence is complete. The Argyll-Robertson pupil is present; there are usually myosis, nyctalopia, and occasionally atrophy of the optic nerve, first manifested by disturbance of the color perception (red-and-green blindness). In the latter condition it has been noted that when blindness has fully developed, the ataxia becomes less pronounced or may disappear completely (*amaurotic tabes*). The visceral crises are characterized by attacks of intense, cramp-like pain, involving usually the stomach, but sometimes affecting the larynx, heart, kidney, bladder, rectum, or other viscera. The laryngeal crises are often accompanied by distressing cough and dyspnea that may simulate the symptoms of aneurism of the arch of the aorta. Trophic changes occur, of which the most common are the arthropathies. These involve particularly the knees, hip-, and shoulder-joints. In addition, the patient may have painless falling-out of the teeth or rapid softening of them. The bones often show a marked tendency to fracture, and an equally remarkable tendency to rapid knitting. Occasionally an osteoarthropathy of the foot gives rise to the so-called tabetic foot, characterized by great thickening of the bones and a real or apparent shortening. In certain cases a painless perforating ulcer develops on the sole of the foot. In the *adynamic* stage of ataxia the loss of muscle-tone has reached so extreme a degree that locomotion is impossible. The patients by this time have usually developed cystitis, and death occurs as a result either of exhaustion or of septicemia.

The Cervical Type of Tabes. This is characterized by the development of the symptoms chiefly in the arms. The leg ataxia, Romberg's symptom, and the absence of knee-jerk may not be present until late in the disease.

Hereditary Ataxia (Friedreich's Ataxia). This is a disease of early life, distinctly hereditary in character, ordinarily affecting several members of the same family. It first appears between the ages of seven and twelve years. It is characterized by incoördination, loss of knee-jerk, weakness, irregular speech, and slight deformities. The first symptom is incoördination of the lower limbs. The muscles grow weaker, the flexors more often giving rise in time

to pes equinovarus with hyperextension of the great toe. The muscles of the back also grow weaker, giving rise to scoliosis; the knee-jerks are absent; the pupillary reflexes remain normal, and intelligence is unaffected. The speech is scanning in character. The gait becomes markedly ataxic, the patients keeping the legs widely separated. In time the paresis and incoördination become so severe that walking is impossible. There are frequently irregular choreiform movements in the muscles and the so-called static ataxia, that is, inability to hold the limb in one position for more than a moment. There is often a nystagmus similar to that observed in multiple sclerosis. Sensation remains normal throughout. The course is progressive.

The *cerebellar* type of hereditary ataxia differs from the foregoing by the fact that the knee-jerks are exaggerated; there is Argyll-Robertson pupil, but no club-foot or scoliosis.

Ataxic Paraplegia.—As the lesions of ataxic paraplegia are found in both posterior and lateral columns the symptoms are those of ataxia associated with exaggerated tendon reflexes. The first symptoms are loss of strength and difficulty in walking. Examination will show distinct incoördination, Romberg's symptom, and exaggerated reflexes. Rigidity and spasticity of the muscles of the legs slowly make their appearance, and ultimately the sphincters may become involved. Dull pains or numbness frequently appear in the extremities, but otherwise sensory as well as ocular symptoms are absent. The upper extremities are also frequently involved.

Spastic Spinal Paralysis (*Lateral Sclerosis*).—This is characterized by weakness of the legs without muscular degeneration and with increased reflexes. The first symptom is weakness or a feeling of heaviness in the legs; then spontaneous cramps appear. The reflexes are greatly exaggerated, and the muscle-tone is so increased, particularly in the extensor muscle of the thigh and the muscles of the calf, that the patient walks with the leg partially extended, dragging the toe along the ground; the arms are rarely involved. The electrical reactions of the muscles are normal. The sphincters, and sensation usually, are unimpaired. If cramps are frequent, however, the muscles may be sore. The adductors may become stronger than the abductors, and a peculiar, cross-legged gait is thereby produced. The disease may occur in several members of the same family (familial form), the symptoms usually appearing in early adult life.

Syringomyelia.—The first obvious symptoms rarely occur before early adult life. The occurrence together of the following group of symptoms is almost pathognomonic: (1) dissociation of sensation; pain and temperature senses are lost, but tactile and muscular senses are retained; (2) degenerative atrophy of the muscles, associated with fibrillary twitchings and alteration of the electrical reactions; (3) trophic lesions which may involve the skin, particularly that of the fingers or the joints. The size and position of the cavity and the extent of the destruction of the substance of the cord by the gliomatosis vary

greatly, and, as a result, the symptoms, which are due entirely to the interference with the structures of the spinal cord, differ considerably in different cases. This difference, however, is quantitative rather than qualitative, and the three groups of symptoms are practically always present. The disease appears to develop with extreme slowness. The earliest symptom may be the occurrence of painless whitlows. Later, muscular atrophies appear. These involve particularly the muscles of the shoulder or the hand. In the latter situation they may give rise to the appearance that occurs in progressive spinal muscular atrophy. At the same time the sensory disturbances become more pronounced, gradually ascending the arm and perhaps involving the trunk. The trophic changes may then assume a more severe form, giving rise to deep, painless ulcerations in the fingers, and perhaps loss of the terminal phalanges. It is rare for the two sides to be affected equally. At times the lower portion of the cord is particularly affected, and then sensory and trophic changes are found in the legs. Ultimately the patient develops scoliosis; trophic changes affect parts other than the hands, giving rise to arthropathies or to a form of dry arthritis with absorption of the bone. There may be vasomotor disturbances, and in some cases inequality of the pupils. The intellect is undisturbed. The patients ordinarily die as a result of exhaustion or pulmonary involvement, but occasionally in the later stages of the disease, bulbar symptoms occur.

Moreau's disease, characterized by the appearance of painless whitlows in the fingers, sometimes associated with deep ulcerations of the soft parts and other trophic changes, is probably only a manifestation of syringomyelia.

Progressive Spinal Muscular Atrophy (Type of Duchenne-Aran).—Chronic Anterior Poliomyelitis.—A chronic progressive degenerative process of the spinal cord, involving chiefly the anterior horn cells. The idiopathic form of the disease usually commences about middle life. The hereditary form appears, as a rule, somewhat earlier. The general course is as follows: The first changes are observed in the muscles of the hand, particularly in those of the thenar eminences, giving rise to the formation of the *ape-hand*. The interossei and lumbrical muscles are next involved, and the interosseous spaces become deeper, the fingers become gradually weakened, and ultimately are fixed in a semiflexed condition—incomplete *main en griffe*. The muscles show fibrillary twitching and give the reactions of degeneration to the electrical current. The process affects first one hand, usually the right, and then the other. As the disease progresses it next involves the muscles of the shoulder, especially the deltoids, and later the muscles of the upper arm, and then those of the forearm. Finally, the muscles of the trunk and even those of the lower extremities become involved. Sensory disturbances are rarely present. Occasionally the patient complains of slight pains and paresthesia. The reflexes are lost early. The course of the disease is exceedingly slow. Emaciation becom-

extreme, but total paralysis occurs only very late in the disease. Death ultimately results from the involvement of the intercostal muscles and the diaphragm, giving rise to respiratory failure. It is usually due to a terminal pneumonia. In addition to the typical course described, the disease may assume many other forms, and sometimes for long periods appears to be arrested.

Progressive Neural Muscular Atrophy (*Charcot-Marie-Hoffman Type, Peroneal Type of Gowers*).—This disease commences about the time of puberty. The muscles first affected are those of the feet and hands. The affected muscles show distinct fibrillary twitchings, and usually the characteristic reactions of degeneration to the electrical current. These reactions of degeneration are also present in the nerves. There is usually a coarse, irregular tremor, and the atrophy of some of the muscles with contractors of others gives rise to various deformities, such as the *ape-hand*, the *main en griffe*, or, if the foot is first affected, to *foot-drop*. Later the foot assumes the position of equinovalgus or equinovarus. In this disease there is sometimes involvement of the sensory fibers, but the nerve trunks are not sensitive. Hypesthesia is also occasionally present. The process usually is restricted to the limbs, the muscles of the trunk and face escaping; bulbar symptoms are almost unknown and the functions of the bladder and rectum are not disturbed. In a form of this disease described by Déjérine under the title of *Infantile Hypertrophic and Progressive Interstitial Neuritis* there are, in addition to the above changes, the symptoms of locomotor ataxia, that is, Romberg's symptom, lancinating pains, ataxic gait, and even disturbance of the pupillary reflexes. The nerve trunks become enlarged and can be felt beneath the skin.

Amyotrophic Lateral Sclerosis.—The disease usually occurs in adults about middle life. In very rare cases it occurs in children. It is characterized by a spastic paraplegia, with exaggeration of the reflexes and degeneration of the muscles. Spasms are common, and may give rise to muscular pains. The symptoms consist of weakness in the legs, which at the same time become stiff; the muscles rapidly atrophy; there are fibrillary twitchings and reactions of degeneration. The arms are usually involved first, the degeneration commencing in the muscles of the hands, and giving rise ultimately to the production of various deformities, such as the claw-hand. The tendon reflexes are greatly exaggerated; there are patellar and ankle-clonus and the Babinski phenomenon. The sphincters are rarely involved, the pupillary reflexes are normal, and there are no sensory disturbances. Bulbar symptoms, that is, paralysis of the larynx, pharynx, and palate, occur still later in the disease. The face has a mask-like appearance, and often saliva dribbles from the mouth. In this stage inspiration pneumonia is quite common and usually causes death.

Bulbar Paralysis.—Bulbar palsy is a paralysis of the motor nuclei in the medulla. It is characterized by the degeneration of the muscles of the lips, tongue, and pharynx, which grow smaller, exhibit fibrillary

Spina Bifida.—Congenital failure of the vertebral arches to coalesce results in extrusion of the dura with or without a part of the cord (meningomyelocele and meningocele respectively). The pedunculated or sessile tumor is usually in the lower portion of the spine and is at first symptomless, later pressure symptoms, such as paraplegia, anesthesia, trophic disturbances, club-feet, and sphincter trouble develop. Pressure on the tumor will usually cause expansion of the fontanelles. Early and marked pressure symptoms are indicative of meningomyelocele.

DISEASES OF THE BRAIN

Affections of the Cerebral Membranes.—External Pachymeningitis.—

A rare condition, usually secondary to traumatism or abscess, characterized by fever, headache, often sharply localized, and convulsions. Frequently the symptoms are masked by the existence of some graver conditions, such as status epilepticus, coma, or uremia. Occasionally no symptoms are produced. If there is much thickening of the membrane, evidence of focal disease in the form of paralysis or convulsion of the Jacksonian type may be present. *Internal hemorrhagic pachymeningitis* is a condition usually occurring in cases of chronic disease. There may be slight fever and headache without other symptoms. In some cases, however, the onset is sudden and apoplectiform in type. The patients develop hemiplegia, unconsciousness, and occasionally unilateral convulsions.

Leptomeningitis.—There are many varieties of this: thus, according to the location, there may be meningitis of the convexity, or of the base; according to the cause, there may be suppurative, epidemic, tuberculous, or serous meningitis.

Suppurative Meningitis.—The patient may complain of malaise and headache for a few days preceding an attack; then there is often a chill, followed by fever, convulsions, and delirium. The headache becomes more intense, and frequently there is projectile vomiting, sometimes with nausea. The headache is often localized in the frontal or occipital regions; occasionally, however, it is general. From time to time there are acute exacerbations, causing the patient, especially if a child, to cry out—the *hydrocephalic* cry.

The skin is hyperesthetic; all the sensory nerves have their functions increased; there are photophobia and inability to tolerate noises. When the skin is scratched a red line appears (*tâche cérébrale*), which is, however, of little diagnostic import. The patient lies with the head drawn far back and the muscles of the neck and rigid. This, however, occurs only when the cervical part of the spinal cord is also involved. Any attempt to straighten the head causes intense pain. Examination of the eye-grounds usually shows intense congestion of the optic nerves, and more or less perineuritis. Sometimes there is also papilledema. The pupils are often unequal.

strabismus and even nystagmus frequently occur. Paralysis of any of the cranial nerves may occur, but is not so common as in tuberculous meningitis. The oculomotor or some of its branches is most frequently involved, although slight paresis of the facial nerve is not uncommon. Fever, headache, and delirium usually persist throughout the course of the disease; the fever is often very high. In the early stages the pulse is usually relatively slow; later it may become rapid. The blood-pressure is always increased. Meningitis due to pyogenic microorganisms, such as the pneumococcus, staphylococcus, etc., may be suspected; when the fever is high; when there is marked retraction of the head, indicating spinal involvement; when there is a leukocytosis; and especially when there is or recently has been some other focus of pyogenic infection in the body, such as pneumonia, sepsis, or middle-ear disease. The course is progressive and almost invariably ends in death. But remissions may occur or there may be considerable temporary improvement after spinal puncture, which is invaluable for diagnostic purposes.

Epidemic cerebrospinal meningitis may exactly simulate the symptoms of purulent meningitis. (See page 499.)

Tuberculous meningitis is the most common form. It affects chiefly the base of the brain. There is usually recognizable tuberculosis of some other part of the body, and often a history of injury to the head. The course is slow, although in rare instances it may be very rapid; the prodromal stage is prolonged and the symptoms are slight, consisting of headache, insomnia, malaise, loss of appetite, progressive emaciation, and occasionally distinct psychical disturbance, such as extreme irritability, light coma, a confusional state, or loss of interest in the surroundings. As the disease progresses, more distinct lesions occur; the cranial nerves are involved; and there may be ptosis, diplopia, or paralysis of the pupil. Paresis or even paralysis of the facial nerve is common, and lesions of the other nerves may develop. Occasionally monoplegia or hemiplegia occurs. The fever is moderate, the course deliberate, and extreme intracerebral pressure develops rather late, so that the blood-pressure may not be greatly elevated. There is moderate leukocytosis (10,000 to 15,000). Spinal symptoms, such as rigidity of the spine and retraction of the head, are comparatively rare. Aside from the discovery of tubercle bacilli in the spinal fluid, the only pathognomonic sign is the presence of tubercles upon the choroid. In very rare instances a secondary infection with some other form of microorganisms occurs in tuberculous meningitis. This appears to render the course shorter and more violent.

Serous meningitis is characterized by a marked increase to the intracerebral pressure, and the symptoms that result from it—headache, coma, or convulsions. There are usually slight fever, moderate leukocytosis, increase in the reflexes, and slight rigidity of the back of the neck. The face is flushed, but eruptions are rare. The diagnosis is usually made by spinal puncture. The fluid is clear, sterile, and under

great pressure. This condition occurs in the course of some infectious process or may be the end-result of prolonged drinking bouts.

Meningismus is a term applied to the meningeal irritation that occasionally occurs in the acute infections that attack children.

Cerebral Vascular Disturbances.—Hyperemia of the Brain.—This may be *active*, the result of alcoholism and other intoxications, acute infectious disease, mental strain and excitement; or *passive*, the result either of general venous engorgement, or local engorgement from venous or sinus thrombosis, tumors or abscesses of the brain, or from obstruction of the circulation of the neck. The symptoms are headache, feeling of fulness and pulsation, vertigo, tinnitus, insomnia, and perhaps delirium. The symptoms are often obscured by the causative condition, and in active hyperemia are more pronounced and sudden in onset than in passive congestion.

Anemia of the Brain.—This may be the result of any severe anemia or of any condition lowering systemic blood-pressure. The symptoms include dizziness, tinnitus, nausea, headache, and transitory loss of consciousness (fainting), which may persist and result in death.

Edema of the Brain.—Passive congestion, chronic nephritis, concussion, abscesses or tumors, and obstruction of a sinus may cause a general or local excess of moisture of the cerebral substance; or an increase in the cerebrospinal fluid may cause cerebral edema, resulting in disordered cerebration, convulsions, paralysis, etc. The symptoms are usually due to the associated conditions rather than to the edema itself.

Cerebral Hemorrhage (Apoplexy).—This usually occurs in advanced life in patients who have pronounced arteriosclerosis. Occasionally, however, it occurs in young syphilitic adults and in children. It is characterized by a great variety of symptoms, depending largely upon the location of the lesion. They may be divided into those of the attack and those of the post-apoplectic stage. The symptoms of the attack consist of prodromata, that is, headache, tendency to vertigo, a sense of fulness in the head, roaring in the ears, perhaps some thickness of speech, and general paresthesias. They may pass off without further disturbance or may lead directly to an attack. The latter is usually characterized by the sudden occurrence of complete unconsciousness, the apoplectic "stroke." The patient falls to the ground, and there is at first a temporary pallor. This is succeeded by flushing of the face, which may become almost purple. The pulse is full, bounding, and compressed with difficulty. The blood-pressure is greatly increased. The breathing is stertorous, slow, and occasionally of the Cheyne-Stokes type; the pupils are usually contracted and often unequal. Often there is vomiting or involuntary micturition or defecation. If, as is commonly the case, the hemorrhage has involved the motor tract, there is complete flaccid paralysis (occasionally rigidity) of one side, with lost reflexes. If death does not occur in the course of the first twenty-four hours, the patient usually begins to show signs of

consciousness, and may be aroused from his comatose condition by sharp questioning. He then may pass into a still more deeply comatose condition, with rise of temperature, followed by death, or there may be no further indications of hemorrhage, and recovery may set in. As a rule, in those cases in which the prognosis is favorable no rise of temperature occurs. It may now be found that the patient has hemianopsia, usually the visual fields on the same side of the lesion being blinded. Conjugate deviation may or may not have existed from the first, the patient ordinarily looking toward the sound side. If the speech centre has been involved, there is absolute aphasia; but even when it is not directly affected, partial aphasia is very common. The hemiplegic limbs remain paralyzed, the others regain their power.

It is now necessary to determine the extent of the damage and to locate as nearly as possible the position of the lesion. Complete hemiplegia may involve the lower branch of the facial nerve, the arm, the leg. The upper branch of the facial and the muscles of the trunk commonly escape, although the former may show slight paresis. Often there is considerable inequality in the paralysis of the different parts. The arm may be more involved than the leg, or the reverse, or the face may escape almost entirely. Sensory disturbances are usually not present. There is sometimes disturbance of only the tactile or the muscular sense. Occasionally, when tactile sense is preserved, there may be loss of the stereognostic sense. Complete hemiplegia with disturbance of sensation almost invariably indicates destruction of the internal capsule upon the opposite side. Motor disturbances in the form of clonic convulsions may also occur in the paralyzed limbs. As the case progresses there are usually more or less return of motor power and almost complete return of sensation. This may, however, be exceedingly gradual, several weeks elapsing before the sensory disturbances have entirely disappeared. The muscles that remain permanently paralyzed gradually atrophy, but nearly always give normal qualitative electrical reactions until the muscular substance disappears, leaving contracted fibrous tissue. The muscles themselves may contract early, the flexors ordinarily overcoming the extensors. Repeated attacks of apoplexy are by no means uncommon, and the double lesions thus produced may give rise to very complex symptom-groups. Hemorrhage into the pons or medulla gives rise to various forms of crossed paralysis. (See also Cerebral Localization and Aphasia.)

In the *ingravescent* form of apoplexy, the hemorrhage is gradual and the symptoms are slower in onset. There may be headache and vertigo, followed by a gradual loss of power in the arm and leg, which in a day or two may be followed by unconsciousness.

Cerebral Embolism and Thrombosis (*Softening of the Brain*).—Embolism of an artery may occur at any period of life, and is nearly always associated with some valvular disease of the heart. An extreme cachexia or anemia, cerebral arteriosclerosis (usually syphilitic), and a weakened circulation predispose to thrombosis.

Embolism is characterized by symptoms very similar to those of cerebral hemorrhage. Prodromal symptoms, in the form of headache, vertigo, weakness, and malaise, are often present. At times there may be also slight impairment of speech, or the patient may be dull and apathetic. The attack usually comes on more gradually than hemorrhage, although this is not invariably the case. In some instances consciousness is not entirely lost, and as a result the hemiplegia may develop before the coma. When unconsciousness does occur, there are usually less congestion of the face and not such marked evidence of increased arterial tension as we find in hemorrhage. Among the other general symptoms may be mentioned convulsions, often epileptiform in type, vomiting, and occasionally delirium. The permanent symptoms resemble exactly those produced by hemorrhage, but recovery is usually more rapid and more complete than in the former condition. As the Sylvian artery is most frequently affected, the symptoms resemble the cortical type of apoplexy.

Thrombosis of an artery may cause similar symptoms or the symptoms may develop gradually. At times it may be preceded by prodromes, as temporary loss of memory, inability to speak or to move certain muscles. However, extensive areas of softening may be found post-mortem which have caused no special symptoms.

Thrombosis of the Cerebral Sinuses.—This may be primary, occurring in anemic, cachectic, and wasting diseases of children as a rule, or secondary to otitis media and mastoiditis, to fracture of the skull, or brain abscess. Primary thrombosis may be obscured by the causative condition, or there may be headache, nausea and vomiting, mental dulness, convulsions, and coma. Secondary thrombosis is a septic condition, and is associated with chills, extremely irregular fever, nausea and vomiting, headache, later with the general symptoms of meningitis.

Cerebral Paralysis of Children.—The palsies of early childhood are the results of injury of the motor centres or tracts, during the intra-uterine life, in the course of a difficult labor, or in postnatal life from trauma, the infectious diseases, etc. They may be *hemiplegic*, *diplegic*, or *paraplegic*. These paralyzes may be sudden in onset, attended by convulsions and unconsciousness; they may be gradual, appearing shortly after birth in those cases due to birth injuries; or they may not appear until the child should start to walk. The paralyzes affect the legs most markedly as a rule and the face usually escapes. They are spastic in type and often contractures result which cause various deformities. Atrophy is rare and the reactions of degeneration are absent. The reflexes are exaggerated and bilateral athetoid movements and chorea are common. Sensation is unimpaired; epilepsy is common; and mentality is impaired.

Little's disease is a form of infantile palsy occurring in children born prematurely, and is the result, supposedly, of arrested development of the pyramidal tracts. The paralyzes are diplegic and paraplegic, while cerebral manifestations are mild.

Amaurotic family history is a condition of idiocy developing a few months after birth, accompanied by spastic paralysis of the greater part of the body, blindness, and marasmus. The disease seems to be familiar and usually ends fatally in early childhood.

Acute Encephalitis.—This is a condition that rarely can be diagnosed during life. It may be suspected, however, if, in the course of some acute infectious disease, the patient develops intense headache, severe delirium, and perhaps local palsies.

Abscess of the Brain.—This is usually secondary to some local focus of suppuration or to pyemia; there is often a history of mastoid disease. It may be acute or chronic. Acute abscesses are characterized by violent general disturbances, chiefly fever, chills, leukocytosis, headache, delirium, and coma. Chronic abscesses give rise to pressure symptoms of local disease, which depend, of course, upon the location of the abscess. The course is usually intermittently progressive; thus there may be first a monoplegia, then hemiplegia, and finally aphasia; or the earliest symptoms may be hemianopsia, followed by paralysis. The commonest seat is in the temporosphenoidal lobe, as a result of infection following ear disease, and often gives rise to mind-blindness or amnesia. Sometimes there are no localizing symptoms if the abscess is located in the so-called blind regions of the brain. Headache, vomiting, disturbance of cerebration, and optic neuritis are usually noted in either an acute or chronic abscess (sooner or later), no matter where localized.

Tumors of the Brain.—Like the preceding lesion, these give rise to two groups of symptoms: general, which are merely those of increased intracranial pressure; or local, which are due to involvement of centres and tracts. The general symptoms of brain tumor are (1) headache; this is usually very severe, of a boring character, and subject to exacerbations; (2) vomiting; this is paroxysmal, and often occurs without nausea; (3) optic neuritis; it usually occurs early, is intense, and often leads rapidly to blindness. The focal symptoms are, of course, numerous. Tumors in the *frontal lobe* may give rise to none or only to slight disturbance of intelligence. The headache is usually frontal, and occasionally in subcortical tumors there may be nystagmus. Tumors in the *motor region* may cause irritative or destructive changes in the tissue. Irritation is manifested by local spasms which may or may not be succeeded by general convulsions (Jacksonian epilepsy). Paralytic lesions belong to the monoplegic or hemiplegic types. Tumors in the *parietal lobe* may cause interference with the muscle sense or some disturbance of vision- or speech-centre, according to their situation. The loss of the stereognostic sense is a common symptom. Tumors in the *occipital lobe* usually cause mind-blindness, that is, inability to recognize objects, and preservation of the pupillary reflexes. If unilateral, they often cause hemianopsia. Tumors in the *cerebellum* usually produce marked disturbance in coördination, and the patient exhibits the peculiar staggering gait, not increased by closing the eyes, that has

been described. Optic neuritis is an exceedingly common complication, but there may be descending atrophy of the optic nerve without preceding choked disk. The headache is severe; it is situated in the posterior portion of the head; there is often tenderness over the external occipital protuberance. The patient may suffer from vertigo, which at times is almost constant. The general symptoms are usually pronounced. There are convulsions, vomiting, disturbances of respiration, and sometimes marked slowing of the pulse. Tumors of the *basal ganglia* produce very variable symptoms, the majority apparently being the result of pressure upon the internal capsule. Tumors of the *thalamus* often give rise to hemianesthesia with loss of mimicry, and in some cases apparently to athetoid movements. In all forms of cerebral tumors, but particularly in slowly growing tumors of the cerebellum, the early symptoms may be those of neurasthenia or even hysteria, and the diagnosis for a long time is exceedingly difficult. Tumors in the different fossæ of the skull often give rise to symptoms dependent upon pressure upon the cranial nerves. In the *anterior fossa* there may be loss of the power to smell upon one side. In the *middle fossa* the nerves chiefly affected are the optic, giving rise to unilateral blindness; or, if the tumor involve the chiasm, to bitemporal hemianopsia; if it presses upon the oculomotor nerve, the abducens, and the pathetic, it may give rise to more or less complete ophthalmoplegia. Tumors in the *posterior fossa* commonly involve the facial and auditory nerves; and facial paralysis with nerve deafness on the same side is characteristic of tumor in this situation. The hypoglossal nerve may also be involved. Tumors may grow slowly, or cease to increase in size, and the symptoms show a corresponding rate of development. In rapidly growing tumors apoplectiform attacks are frequent, but a certain amount of compensation occurs, and remissions are not uncommon. In slowly growing tumors the symptoms may remain apparently stationary for long periods. Tumors are sometimes entirely latent, and are only discovered accidentally at the autopsy.

Hydrocephalus.—Hydrocephalus may be congenital or acquired. It is characterized by an extraordinary alteration in the contour of the head, which becomes greatly enlarged and globular in shape, while the face remains small and infantile in appearance (excepting the acquired form which develops in later life and which causes no change in the shape of the head and the symptoms of which are referable to the causative condition and which usually simulate those of a tumor). The fontanelles and sutures remain widely open, or are closed only very late by Wormian bones. The veins of the scalp are very distinct, and occasionally fluctuation may be detected in the head. The symptoms are sometimes exceedingly pronounced; at other times entirely absent. Persons with a moderate degree of hydrocephalus have displayed through life a normal intelligence. In pronounced cases the head is heavy and the muscles of the neck are unable to support it. The child develops slowly in intelligence or may even be an imbecile or an idiot, and epileptic convulsions

are very common. Occasionally ocular symptoms may be present. These consist of ptosis, strabismus, or nystagmus, and sometimes of atrophy of the optic nerve and blindness. Pain, shown by the hydrocephalic cry, is quite common.

Multiple Sclerosis.—This is a condition that involves the spinal cord and brain. The situation of the lesions is not constant, and therefore the symptomatology is variable. It usually commences at about the age of thirty years. The characteristic symptoms are intention tremor, nystagmus, and scanning speech. The *intention tremor* involves chiefly the limbs, and causes disturbance of writing, walking, etc. It is most easily elicited by having the patient grasp some small object. There may also be rhythmical oscillations of the head. There is usually persistent lateral nystagmus. The tendon-reflexes are greatly exaggerated, and there are ankle-clonus and spastic gait. The pupillary reactions are normal. Occasionally diplopia occurs as a result of abducens paralysis; it may be transient. In a large proportion of the cases there is more or less complete atrophy of the optic nerve. The speech is slow, drawling, and often tremulous. The voice lacks expression, and, on account of a slight nasal tendency, is usually disagreeable. Not infrequently there may be stuttering. Sensory changes are rare. Less frequent symptoms are vertigo, occurring in paroxysmal attacks, diminution of intelligence, and alternate states of depression and exaltation. Apoplectiform and epileptiform attacks may also occur. The disease is usually chronic, but from time to time there are exacerbations.

General Paralysis of the Insane.—General Paresis—Dementia Paralytica.

—A form of progressive dementia characterized by delusions of grandeur, or states of depression associated with exacerbations of maniacal character. There are, in addition, weakness and tremors of the muscles of the face, paresis of the extremities, Argyll-Robertson pupil, and peculiar disturbances of speech. It is a disease of middle adult life, the first symptoms occurring between the thirtieth and fortieth years. There is often a history of syphilis. It is usual to recognize three stages: (1) the prodromal stage, characterized by irritability or sometimes by depression; (2) diminution or loss of the moral sense; and (3) impaired judgment and a tendency to extravagance and dissipation. Frequently symptoms associated with degeneration, such as intolerance for alcohol, intense egotism, etc., appear. The sexual appetite in this stage is often greatly increased. Memory fails, and the intellectual capacity is considerably diminished. There are often slight disturbances of speech, and sometimes paralytic pupils. Frequently there are insomnia and occasional attacks of migraine. In the second stage, which usually develops gradually, the attacks of migraine are replaced by apoplectic or epileptic attacks, or by distinct maniacal conditions; memory is greatly impaired, the intellect is considerably disturbed, the patient becoming unable to do easy mathematical problems, to comprehend his environment, or to sustain a simple conversation. Usually there

are delusions of grandeur, the patient believing himself rich, beautiful, successful, intelligent, and reiterating constantly his advantages, although from time to time there are states of depression and partial recognition of the failure of power. In other cases, however, particularly among chronic alcoholics, there is distinct melancholia, the patient is hypochondriacal, or may have delusions of persecution or a sense of misfortune. The disturbances of speech are characteristic; the most common is the omission of syllables. This may best be tested by asking the patient to repeat certain words, particularly those containing a number of r's and l's, as "third artillery brigade," "truly rural," etc. The intonation is often monotonous, and often there is an unpleasant nasal drawl. There is marked tremor of the lips and of the tongue, producing a sort of ataxia in the speech, with the disturbances of the formation of nearly all the sounds. The pupillary changes are similar to those described in the prodromal stage, but usually more pronounced. Piltz and Westphal have described a peculiar reflex contracture of the pupil upon forcible closure of the lids that is more common in general paresis than in any other disease. The extremities are weak, and often exhibit distinct tremors. Trophic lesions are not so common as in tabes, but do occur. They are of the same character and possibly due to tabetic changes in the cord. Finally, the patient becomes completely demented, usually lies quietly and placidly in bed, or occasionally mutters unintelligible sounds. Sensation, either as a result of impaired perception or because of degenerative changes in the peripheral nervous system or the spinal cord, becomes greatly impaired, particularly the pain sense. The patient is unable to stand, and has involuntary or rather unperceived micturition and defecation, and frequently develops bed-sores or cystitis; even rupture of the bladder may occur. A curious and quite common symptom is the gnashing of the teeth, which in some cases is almost persistent. Death usually occurs from exhaustion. Among the less frequent symptoms are a curious unsteadiness of gait, exaggeration of the reflexes, and rapid diminution in weight, particularly in the last two stages.

The *laboratory examinations* are of particular value in diagnosing the disorder. The Wassermann reaction, as in tabes, is practically always positive. Noguchi's butyric acid reaction is likewise positive, and a count of the leukocytes of cerebrospinal fluid constantly shows a lymphocytosis while the fluid contains much albumin.

Syphilis of the Nervous System.—This occurs most frequently in men between twenty and forty years of age and usually appears some three to ten years after the primary lesion. The symptoms are usually extremely irregular and the diagnosis is commonly based upon these irregular nervous symptoms associated with a luetic history or a positive Wassermann reaction.

Syphilis of the Brain.—The brain lesions may be (1) syphilitic arteriosclerosis—resulting in aneurism and subsequent rupture of a cerebral bloodvessel (apoplexy) or thrombosis and subsequent softening;

(2) gumma (a) single—resulting in the general and focal symptoms of a brain tumor; (b) multiple—usually causing meningeal symptoms (headache, insomnia, vertigo, and stupor), irregular symptoms of encephalitis (convulsions, notably) and local symptoms, the result of involvement of the cranial nerves (particularly the second, third, fourth, and sixth or possibly other centres); any one of these symptom-groups may overshadow the others; (c) gummatous meningitis—caused by microscopic gummatous formations and resulting in symptoms very similar to those caused by multiple macroscopic gummata involving the meninges.

Syphilis of the Spinal Cord.—The symptoms of spinal syphilis are usually associated with those of cerebral syphilis. They may be those of arteritis with secondary softening, of meningitis with secondary cord changes, of myelitis and secondary sclerosis, or of tumor formation from large gumma of the cord or meninges.

Parasyphilitic Diseases.—Locomotor ataxia and general paresis are diseases which are in some way dependent upon the toxins of syphilis.

NERVOUS DISEASES OF FUNCTIONAL OR UNKNOWN ORIGIN

Acute Delirium (*Bell's Mania*).—This is a disease characterized by prodromata, a stage of excitation, and usually terminating in death. The prodromata consist of disturbances of the general health, loss of appetite, and insomnia. The patient is restless, anxious, and may show diminution of intelligence, and become more or less violent. He then rapidly passes into the stage of excitation, is restless, noisy, and frequently homicidal. Sometimes there are delusions of persecution, and he attempts to escape. In addition, there is high fever, profound prostration, dry tongue, and rapid and weak pulse. The patient refuses all food, is continually active, and emaciates very rapidly. Among the objective symptoms are increase of the reflexes, narrowing of the pupils, and hyperesthesia, with more or less hypalgia. From this stage the patient passes into a state of collapse.

Migraine (*Hemicrania*).—This is a disease characterized by paroxysmal attacks of headache associated with nausea and vomiting, and frequently with disturbances of the special senses. The headache is peculiar in that it commences slowly as a dull but severe pain that gradually increases in intensity, with occasional exacerbations or throbbing, and is limited to or greater on one side of the head. The patient experiences a sensation of intense nausea that may be followed by vomiting. There may be photophobia, hyperacusis, and occasionally the appearance of scotoma. Sometimes the patient complains of dimness of vision, and this may affect only part of the visual field. Occasionally there is temporary aphasia, particularly if the pain occurs in the left side of the head. In addition, the patients may observe paresthesias, or occasionally stiffness or spasms in a limb. The paroxysms

usually terminate in prolonged sleep. Sometimes there is a severe attack of polyuria. The intelligence is not impaired.

Epilepsy.—Grand Mal.—This is a condition characterized by attacks of clonic convulsions associated with loss of consciousness and usually some impairment of intelligence. In the characteristic epileptic fit we can usually distinguish two stages: (1) the prodromal stage, and (2) the convulsive, which frequently starts with a cry and is followed by tonic, then clonic spasms, to terminate in coma. In the prodromal stage, varying auræ are usually present. A patient may have either a curious sensation in the epigastrium, paresthesia in a limb, and the subjective sensation of movement, or disturbance of the special senses, particularly an unpleasant odor or a whirring sound. Sometimes the sensations are painful or distressing, as a sense of constriction about the throat. At other times there is giddiness, vertigo, or nausea, or the recurrence of some particular idea. Occasionally the auræ consist of some imperative movement, such as whirling about, running, or jumping. At the commencement of the attack there is usually a cry—the epileptic cry. Ordinarily this is a curious sort of gasping, but in some cases it may be a loud shriek. The patient then falls suddenly to the ground, and the tonic spasm commences. The head and the eyes show conjugate deviation; the face is bluish and pallid; the mouth is filled with frothy fluid, which is often blood-stained, because the tongue has been bitten; and the limbs are in tonic contraction. This is soon replaced by a violent to-and-fro tremor. The patient is completely unconscious. There is no conjunctival reflex; the pupils are widely dilated; frequently the urine is passed during the attack, and there is occasionally profuse sweating. Toward the end the convulsive movements become less frequent and gradually stop. The patient is relaxed and profoundly unconscious. Respiration is reestablished: at first it is irregular, but gradually becomes more and more steady. The cyanosis disappears, and the patient usually passes into a profound sleep. This may last several hours, and he then awakes, feeling dull and fatigued, but otherwise normal. At other times, immediately after the attack, there is vomiting or nausea, and sometimes a feeling of excessive hunger. The attacks may occur with very varying frequency. In some cases the interval may be years, in others months, weeks, or days; or several attacks may occur in the same day. The severity of the case is to be determined rather by the frequency of the attacks than by their individual violence. If they occur so frequently that one is not completed before the other commences, and the patient remains unconscious for some time, the condition is termed *status epilepticus*, in which the pulse and respiration are rapid; hyperpyrexia is common and the patient frequently dies from exhaustion.

Petit Mal.—In this condition the loss of consciousness is so transitory and the motor symptoms are so slight that its nature often escapes detection. The patient, if talking, will suddenly stop for a moment; there is a peculiar rigidity of the expression, and perhaps slight swayin

which will disappear almost immediately. Sometimes after these attacks there will be a feeling or drowsiness for a short period. Auræ may be present in the form of giddiness or twitching of a limb. The attack may also, occasionally, be ushered in with a scream or a peculiar gasping expiration. Immediately after the attack automatic movements may be performed. Attacks of *petit mal* often occur during sleep.

Focal Epilepsy (*Jacksonian Epilepsy*).—This form resembles general epilepsy, with the difference that the motor or the sensory disturbances always commence in the same part of the body, and from this part may gradually extend until they become general. Thus the thumb may be first affected, showing a tonic and then a clonic spasm; then the hand, the arm, and the whole of that side, or both sides; or the disturbances may commence in the foot. In other cases, the spasm may be limited to the muscles in relation to the part in which it started or it may be confined to one side of the body. The disease almost invariably indicates the existence of an irritative focal lesion in the cortical motor area.

Psychical Epileptic Equivalent.—Here the usual seizure is replaced by psychical disturbances in which the patients perform extraordinary acts, such as running about, disrobing and committing violent assaults without subsequently recollecting these acts.

General Symptoms in Epilepsy.—Epileptics are usually dull, apathetic, having a tendency to excess in eating. In many cases there is a distinct mental impairment, or, when the disease occurs early in life, there may be congenital imbecility or idiocy. The temper of epileptics is usually irritable, and they are likely to commit acts of violence. They are peculiarly intolerant to alcohol.

Hysteria.—Hysteria is a disease due to disturbance of the self-control, producing a curious complex of symptoms that appear to be the result of imitation or of a desire to attract attention or sympathy, associated with certain disturbances of the special senses and of sensation. It usually occurs in young adults, especially in women, although males are frequently affected. There is often neuropathic heredity, and frequently the stigmata of degeneration are present. The psychical symptoms are a certain tendency to self-consciousness, so that the patient is anxious to describe his or her sufferings to surrounding persons; is in the habit of performing ludicrous or startling acts for the purpose of attracting attention; is emotional, weeping or laughing readily, and is often irritable and suspicious. Among the *sensory symptoms* are areas of tactile anesthesia or analgesia. These may involve exactly one-half of the body, including the accessible mucous membranes, or they may be symmetrical in distribution on both sides of the median line, and often form geometrical figures. They are not the result, apparently, of simulation on the part of the patient, because they remain unchanged for a number of days. Tenderness, that is, hyperalgesia, may be present over the ovaries and the spine. The areas of anesthesia may be transferred from one part of the body to

the other, either spontaneously or as a result of suggestion. The special senses may have their function exalted, so that the patients have an extraordinary acuteness of smell or hearing, or find it difficult to endure strong lights. Depression of the function of the special senses is perhaps more common, particularly loss of the sense of smell and of taste. Hysterical deafness is exceedingly rare. Hysterical blindness not infrequently occurs, and is characterized by widely dilated pupils which usually react to light, and by normal eye-grounds. The hysterical stigmata associated with the eye are of great importance, partly on account of their peculiarities, partly on account of their persistence. The most frequent is simple contraction of the formed field. This, however, occurs in other conditions, and is therefore not so characteristic as contraction of the formed field with inversion of the color field, that is to say, a red object will be seen farther from the central visual point than a blue one. Monocular diplopia, in the absence of structural defect in the eyeball, is pathognomonic of hysteria. In rare cases three images may be perceived.

The *motor symptoms* are paresis, or occasionally complete paralysis. The commonest form of this is *hysterical aphonia*, in which the patients are unable to contract the vocal cords for the purpose of producing sound, but may be perfectly able to cough or perform any other function with them. In these cases speech usually returns suddenly under the influence of a strong emotion or suggestion. The paralysis in other parts of the body occurs in imitation of some form of organic disease. Thus there may be paraplegia, hemiplegia, or monoplegia. Loss of power is rarely complete. The electrical reactions remain normal, although the degree of resistance in the skin may be greatly increased. The reflexes are exaggerated, especially those due to cutaneous irritation, such as the plantar reflex, but ankle-clonus does not occur. The gait may be staggering, imitating cerebellar ataxia or the ataxia due to intoxication; sometimes there are tremors, coarse and irregular, and rarely constant. In some cases of hysteria actual contractures of the muscles occur. Spasmodic contractions sometimes occur in the muscles of the abdomen, giving rise to an apparent or *hysterical abdominal tumor*. Actual trophic changes may also occur in hysterical patients, but these are rare in this country. There may be hemorrhages into or from the skin, particularly from the forehead, palms of the hands, and soles of the feet, or there may be localized areas of gangrene of the skin. Paroxysmal seizures may occur which may be divided into the prodromal period and the convulsive. The *auræ* consist of a variety of sensory disturbances, of which the most common is the sensation of a ball rising in the throat (*globus hystericus*). The patient may also have a sensation of heat or cold, or moisture of the skin or various painful impressions. It is impossible to describe all the movements that occur in the *grande crise*. The convulsion may be tonic or clonic. The patient may assume the most extraordinary positions. Among the most characteristic is opisthotonos, in which the heels and back of t

head rest upon the floor or bed, while the body forms an arch; or the patient may assume attitudes that suggest or are characteristic of mirth, sorrow, fear, passion, etc. *Catalepsy*, a condition in which the limbs are plastic and remain in any position in which they are placed, may supervene. Consciousness is rarely entirely lost, although there may be subsequent total amnesia for the period of the attack, and no matter how violent the movements of the patient, injury to any part never occurs. Gradually the movements become less violent, the patient becomes quiet, and consciousness returns. During the attack the pupils are usually dilated, the reflexes may be increased, and respirations are commonly extremely rapid. After the attack the patient may be perfectly normal. At times there may be persistent, perverse tendencies, such as unwillingness to eat, or at least a simulation of fasting.

Neurasthenia.—Neurasthenia is a disease characterized by an exceedingly complex symptomatology. The most common general symptom is a subjective sense of fatigue, both mental and physical. The symptoms may be divided into the general and special groups: the former including those common to all forms of neurasthenia, the latter those associated particularly with subjective and objective functional disturbance of the various organs. The mental symptoms are various. The patients are usually querulous, depressed, and hypochondriacal. They are very irritable, but incapable of prolonged emotional exaltation. They find difficulty in concentrating their attention, particularly upon those subjects with which they have previously been familiar. Memory is impaired, and the intellectual capacities are apparently diminished. An important symptom is the insomnia. This may be of all varieties, but ordinarily the patient, after sleeping in the early part of the night, will awaken and be unable to sleep again for some hours. Frequently they complain of unpleasant or frightful dreams. Among the sensory symptoms the most important is headache. This is of a peculiar but almost typical form. The patient complains of a heavy, dull feeling, as if wearing some heavy object. Occasionally the pain is localized—sometimes to the occipital region and sometimes to a circumscribed area. Another symptom that is very common is pain in the back. This is usually felt in the neck or in the lumbar and sacral regions; it is of a dull, persistent character, and may be associated with points of tenderness over the spine. Occasionally there are disturbances of the special senses. The patient may complain of inability to see sharply or there may be *muscae volitantes*. At other times he will fail to hear distinctly, or may complain of roaring or tinnitus. Actual diminution of the visual power or of the sense of hearing does not occur. The patients may complain, however, of paresthesias in the limbs and of various symptoms, usually the result of suggestion. Sensation is otherwise normal. There is usually a general decrease in muscular power. Sometimes this may be preserved for short periods of activity, but fatigue, as a rule, comes

on very rapidly. At other times it is impossible for the patient to exert the amount of force that would be normal for his muscular development. A fine tremor of the extremities is often visible. This may be persistent or readily exhausted; in addition, fibrillary twittings of the muscles not infrequently occur. The tendon reflexes are generally exaggerated. Absence of the knee-jerk does not occur in neurasthenia. The cutaneous reflexes are sometimes greatly exaggerated, sometimes decreased. Vaso-motor symptoms are very common. The patient flushes easily, and there is often *dermographia*; he complains of palpitation and occasionally of irregularity of the heart's action. Tachycardia is not uncommon. Often perspiration is produced by slight exertion.

In addition to these symptoms, the neurasthenic may complain of various local disorders of the nervous system. Another common form is gastro-intestinal neurasthenia. The patient may complain of excessive acidity, and, in fact, vomit from time to time masses of acid material. Constipation is an exceedingly frequent symptom. Finally, the patient may be a sexual neurasthenic and believe himself suffering from organic or functional disease of the genital organs. The degree of neurasthenia is spoken of as mild or severe, according as the symptoms are slight or pronounced.

Traumatic Neurosis (Railway Spine).—This usually follows as a result of shock from a railroad or some other severe accident. The usual course of these cases is as follows: After some severe accident, with or without demonstrable injury, usually of such a nature that a claim for damages can be made, the patient begins to complain of various paresthesias in the supposedly injured part, usually the back; these gradually increase in severity, may be associated with motor disturbances, such as tremor, spasms, or paresis, occasionally with objective sensory disturbances, or any of the numerous symptoms of neurasthenia. The patient devotes himself to the study of his symptoms, to the exclusion of other forms of mental occupation. The course is variable, but in the great majority of cases more or less improvement occurs as soon as the damages have been obtained. Nevertheless, some cases do not improve under these circumstances, and others occur under circumstances that preclude any possibility of recompense. The diagnosis should never be made until the patient has been under skilled observation for a considerable period.

Occupation Neuroses.—These are characterized by the development of pain in the limb employed when the attempt is made to perform some habitual movement. They ordinarily occur in early adult life, particularly in neurotic individuals. The subject has often been in the habit of performing the motions that cause the trouble in a manner. In *writers' cramp* he first notices that he becomes readily fatigued than usual, and there may be dull pains in the wrist or in the palm of the hand. The painful sensations may then creep up the arm, often as far as the shoulder. They are rarely severe by their persistent, dull character are extremely annoying. The

symptoms are characterized by incoördination and tremor at times and by a tonic spasm of the muscles employed in grasping the pen. The writing is usually heavy and often quite illegible. The electrical reactions are normal or only slightly altered. If the patient learns to write with the left hand, the symptoms of the disease usually develop in it after a short time. Similar symptoms occur in piano-players, dairy-maids, telegraphers, and various other persons who are obliged to perform the same type of movements for long periods.

Acute Chorea (*St. Vitus' Dance*).—Sydenham's chorea is a disease of early childhood and in all probability is infectious in origin. The attacks may recur for a number of years. They are nearly always more severe in winter than at any other season. Girls are affected slightly more frequently than boys. It is characterized by irregular twitching movements affecting various groups of muscles in the body that are usually functionally associated, so that the movements appear to be the result of voluntary innervation. These movements may be generally distributed, are more pronounced on one side than on the other, or may even occur in only one part of the body. They may involve the muscles of the face, the arm, the leg, or the muscles of the trunk, particularly the diaphragm, giving rise to an irregular jerking inspiration. They may vary in severity from slight, imperceptible contractions to severe, general convulsive movements in which the violence is so great that bruises or even fractures may occur. They cease during natural or artificial sleep. As a rule, the affected limbs are slightly weaker, and in some cases this paralysis is very pronounced (*paralytic chorea*). The mind is usually clear, but there may be loss of memory or irritability of temper. In a few cases with violent movements there are pronounced insomnia and violent delirium (*chorea insaniens*). Speech may be affected either as a result of choreic movements of the lips or on account of psychic disturbance. The reflexes, sensation, and electrical reactions of the muscles are normal. Chorea is often associated with articular rheumatism and endocarditis; it occurs largely in certain seasons; occasionally fever occurs; and at times it seems to be mildly contagious; all points in favor of the view that chorea is an infectious disease caused by a specific parasite.

Huntingdon's chorea is characterized by the development between the ages of twenty and forty, of choreiform movements of moderate degree, associated with gradually progressive dementia. The disease is strictly hereditary, occurring only in the offspring of those who have suffered from it; in some families it has been traced through five generations. The twitchings resemble those of chorea, but are rarely violent, and are often associated with a slight rigidity. The earliest mental symptom is usually loss of memory; later, there may be delusions of grandeur or melancholia. Usually life is prolonged to an advanced age, the mental symptoms gradually passing into the type of severe senile dementia. A curious feature is the tendency of the patient to avoid society.

Choreiform Disorders.—In addition to acute chorea, Huntingdon's chorea and paramyoclonus multiplex, which have already been discussed, there are certain other conditions, frequently described as choreic, characterized by irregular, involuntary muscular contractions. They are as follows:

Chorea Electrica.—There are several varieties of this condition—one occurring in adults, characterized by lightning-like contractions of groups of muscles of the neck, sometimes those of the trunk or those of the extremities—so-called (Henoch's chorea); another, *Dubini's disease*, which appears to be an infectious process, commences with violent pains in the head, neck, and back, slight fever, and general convulsions. Death is the usual termination.

Habit Chorea.—Habit spasm is characterized by the repetition of some peculiar, unnecessary movement, such as shrugging the shoulders, winking the eye, rubbing the elbow against the side, etc. Emotional disturbances or the presence of bystanders always increase the symptoms.

Saltatoric Spasm (*Jumpers' Disease*—*Latah*).—Saltatoric spasm is a hysterical manifestation in which the patient, whenever he or she attempts to stand, is compelled to rise on the toes or even to spring from the ground. The spasm disappears if the patient lies down, but may be produced by pressure upon the soles of the feet. It often occurs as an epidemic manifestation, affecting whole communities at once.

General Tic (*Maladie de Gilles de la Tourette*; *Maladie des tics convulsifs*).—General tic is a psychical condition characterized by curious movements of the limbs, grimaces, and the utterance of words that have no relation to the environment, and are often profane or obscene (*coprolalia*) or the imitations of sounds heard (*echolalia*). It usually appears during early adult life, but may develop in childhood. The patient becomes more or less melancholy, and may even be violently insane.

Paralysis Agitans.—This is characterized by a peculiar, fine tremor of the extremities, rigidity of the muscles, a characteristic attitude, disturbance of gait, and gradually progressive paresis. The disease usually occurs late in life. The first symptom noticed is slight rigidity or impairment in agility of the arms. Later this rigidity involves all the muscles of the body, including those of the face, and there is a peculiar rigidity of the facial muscles, causing loss of expression, which is perhaps the most characteristic symptom of the disease. The rigidity also causes the patient to assume a typical attitude: the body and head are thrust forward, and the arms are slightly flexed and swing forward; if the patient stands, the knee and hips are slightly flexed, and the trunk is carried back for the purpose of balancing; the whole attitude, as well as all the movements, indicates stiffness. It will now be found that the patient will have difficulty in rolling over, if lying down, and that there is difficulty in commencing to walk and afterward a tendency to take quick steps (*festination*). The patient watched, will be seen to have from time to time a slight movement forward or back-

ward, which, if he is standing or walking, may cause him to fall in one direction or the other (*propulsion, retropulsion*). Speech is also involved, difficulty in articulation being characterized at first by slight halting and then by the rapid utterance of the words. The tremor usually commences in the hands; it is spoken of as pill-rollers' tremor (*q. v.*). The tremor of the head is a to-and-fro nodding movement. There may also be irregular movements of the toes or legs. The tremor is increased by excitement. In a few cases the tremor is entirely absent or occurs only at intervals (*paralysis agitans sine tremore*). Objective sensory disturbances are rare; the patients usually suffer from a persistent subjective sensation of heat, which is occasionally associated with an actual increase of the surface temperature. The patients are excitable, in spite of the lack of facial expression, and may complain of insomnia. Trophic changes are rare; thickening of the skin is often present, and has been supposed to be the cause of the rigidity; but the skin is sometimes very delicate.

Tetany.—Tetany is probably not a functional disease but a disorder of calcium metabolism due to insufficiency of the parathyroids resulting in hyperexcitability of the nerve cells. It occurs endemically in certain parts of Europe, notably Vienna, and for some reason has a special predilection for shoemakers. Acute infections, certain intoxications, as ergot, chloroform, and lead-poisoning, uremia, gastro-intestinal disorders, such as gastric dilatation, rickets, and pregnancy, and lactation seem to predispose to the disorder. Thyroidectomy occasionally causes tetany, due to the removal of the parathyroids. Tetany is characterized by tetanic spasm of the muscles of the arms and the persistence of peculiar nervous and mental alterations. The attack usually commences with paresthesia or pain in the limbs; then the muscles controlling the fingers become stiff. The flexors gradually contract and draw the fingers and thumb together—the so-called obstetrical hand. The spasm is of tonic character, and may last for several minutes or even for many hours. It is often associated with intense pain. Nearly always it is bilateral. During the interval it may be reproduced by pressure upon the nerve trunks, particularly the median nerve (Trousseau's sign). The muscles show marked irritability to mechanical stimuli, particularly those of the face, and twitching may be caused by tapping upon the trunk of the facial nerve, upon the malar bone, or over the infra-orbital foramen (Chvostek's sign). The muscles show extreme electrical irritability (Erb's sign) and the patient is extremely sensitive to the induced current (Hoffmann's sign). Often there is slight fever. Very rarely trophic changes occur.

Family Periodic Paralysis (Goldflam's Disease).—This is a disease characterized by the occurrence, from time to time, of paralysis of all four extremities. The paralysis is usually flaccid in type, occurs without pain, and is associated with extraordinary increase in the electrical resistance of the skin. The disease usually occurs in several members of the same family, the paroxysms lasting three or four days.

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